ARCHIVES OF SURGERY

VOLUME 41 NOVEMBER 1940 NUMBER 5

COPYRIGHT, 1940, BY THE AMERICAN MEDICAL ASSOCIATION

DISEASES OF THE ESOPHAGUS

ESOPHAGOSCOPIC CONSIDERATIONS

LOUIS H. CLERF, M.D.

PHILADELPHIA

The replacement of inferential diagnostic methods by objective procedures has revolutionized methods of diagnosis and treatment of disease. This is particularly true of diseases of the esophagus. Inspection of the entire length of the esophagus has conclusively demonstrated that many of the inferential diagnoses made in preesophagoscopic days were based largely on error.

To understand more fully the symptoms, diagnosis and treatment of esophageal disease it is important to recall that the esophagus is a long, redundant and movable tube contained in great part in the thoracic cavity, where it occupies whatever space may be allotted it by surrounding viscera. Its fixed points are at the hypopharynx and at the diaphragm. Its course is moderately tortuous, and its lumen presents certain anatomic constrictions, several of which are of clinical importance. The upper end of the esophagus is maintained in a state of tonic closure by the cricopharyngeus muscle, the horizontal portion of the inferior constrictor of the pharynx, and opens only with the passage of fluids or food. At the level of the diaphragm, where the esophagus passes from the thorax into the abdominal cavity, there is a constriction, the hiatus esophageus, which is also referred to as the "diaphragmatic pinchcock." This opens only to allow food or fluids to pass and plays an important part in preventing gastric contents from entering the esophagus. Normally the arch of the aorta and the left main bronchus do not encroach on the esophageal lumen to a degree sufficient to produce interference with function.

The esophagus is an integral part of the alimentary canal, and its function is to transfer foods, fluids and oral secretions from the mouth into the stomach. This is accomplished by a coordinated involuntary action of its musculature and is normally unassociated with subjective sensations. In addition to the normal function of swallowing, the esophagus may empty itself of material contained within its lumen by regurgitation. This must be distinguished from vomiting, since in the

case of regurgitation the expelled materials have not reached the stomach and should therefore contain none of the gastric juices or products of gastric digestion.

SYMPTOMS OF ESOPHAGEAL DISEASE

Dysphagia.—In the presence of disease of the esophagus the act of swallowing usually gives rise to certain subjective sensations which may be indefinite and undescribable or may be sufficiently marked to interfere seriously with esophageal function. This difficulty in swallowing is designated dysphagia and constitutes the most constant and the most important symptom of esophageal disease. Other complaints often considered as a part of the so-called symptom complex of esophageal disease are odynophagia, regurgitation, loss of weight, hematemesis, pain and hoarseness.

Dysphagia, the most important symptom, may vary from a feeling as of a suggestive lump or other curious sensation in the throat to serious interference with the passage of food. No difficulty is encountered in the recognition of dysphagia in a patient whose esophagus is almost completely obstructed and who is no longer able to swallow solid or soft foods and has lost considerable weight. The greatest difficulties lie in the proper interpretation of vague sensations referable to the esophagus and usually associated with the swallowing of food or of saliva. Often the patient can give no clear account of his symptoms, and unless one is sympathetic to these early manifestations of esophageal disease, errors in diagnosis may result. It is important not to ascribe these subjective sensations to a supposedly functional disorder. Cancer of the esophagus often begins with very slight disturbance of the swallowing function. Recognition of this fact probably would result in earlier diagnosis of an otherwise hopeless disease.

Odynophagia.—Odynophagia, or painful swallowing, is not commonly observed as an early symptom of esophageal disease. In certain conditions it is never observed. It is usually indicative of extension of inflammatory or neoplastic infiltration to contiguous structures or viscera. In the cervical portion of the esophagus pain is commonly referred to the level of involvement; in the thoracic portion, however, it is usually referred to the substernal region or may extend to the back. Pain referred to the back is a very important diagnostic sign in cases of suspected foreign bodies in the thoracic portion of the esophagus and in cases of esophageal ulceration. It is frequently without value, however, as a means of localization of disease of the thoracic portion of the esophagus, since the pain areas do not correspond to the location of the lesion.

Regurgitation.—Regurgitation, often confused with vomiting, consists of the ejection of material contained in the esophagus and is due to a reversal of peristaltic action. It is frequently observed in cases of

so-called cardiospasm as well as in all conditions associated with a high degree of esophageal stenosis and represents an attempt on the part of an intolerant esophagus to rid itself of contained foods or secretions which cannot be promptly transported to the stomach. In a study of the ejected material one must recall that it has not been in the stomach and therefore lacks digestive agents and products of digestion.

Loss of Weight.—In cases of disease of the esophagus, loss of weight is primarily the result of progressive starvation and is a late manifestation.

Hematemesis.—The vomited blood may have been swallowed from the upper air or food passages, or it may have had its origin in the duodenum or in the stomach. Much confusion has resulted from attempting to explain the presence of blood on the basis of inferential methods.

Other Symptoms.—Hoarseness, dyspnea and cough are not uncommonly observed with certain diseases of the esophagus. Hoarseness may result either from neoplastic infiltration of the larynx itself or from involvement of a recurrent laryngeal nerve. It is a common symptom of cancer of the cervical part of the esophagus. Dyspnea occurs if there is marked narrowing of the airway either by direct infiltration of the tracheal walls or by compression of its lumen, as in the case of a large pulsion diverticulum of the pharynx. Cough results from overflow of secretions into the larynx and may be observed in any of the conditions which interfere with normal esophageal drainage and in which there is laryngeal involvement. Cough also is an important symptom of esophagotracheal or esophagobronchial fistula.

METHODS OF DIAGNOSIS

A carefully taken history is of importance. Disturbances in swallowing should be investigated with regard to the mode of onset and duration, the types of food producing them, their localization and the character of the sensations of discomfort produced. Regurgitation should be studied in relation to the taking of food. Unfortunately there often is lacking a clear account of the onset of dysphagia. A patient with extensive cancer may state that he has had difficulty in swallowing for many years or that his difficulty has been only of several weeks' duration. In either case there is a discrepancy between the onset of symptoms and the duration and extent of the disease. This presents insurmountable difficulties which cannot be compensated for by any method of diagnosis or treatment and is one of the reasons why so little progress has been made in the treatment of esophageal cancer.

The mouth, throat, larynx, pyriform sinuses and neck should be routinely examined. Accumulation of fluid in a pyriform sinus is a

positive sign of esophageal obstruction. A general examination of the chest should never be omitted. Serologic studies of the blood should be performed. A positive Wassermann reaction and the signs of aneurysm may readily explain symptoms referable to the esophagus.

With the development of roentgenologic and esophagoscopic methods many of the older diagnostic procedures have become obsolete. Auscultatory methods to determine the function of the esophagus are not trustworthy and should have no place in an esophageal study. The use of the bougie to detect esophageal obstruction is not scientific and is extremely dangerous. It is dependent on meeting with an obstruction and therefore is without value in conditions unassociated with obstruction or in the presence of early lesions that have not become obstructive. While the use of the rigid stomach tube is limited, it is of value in the diagnosis of paralysis of the cricopharyngeal muscle and in carrying out esophageal lavage.

The function of the esophagus can be determined by only one method of examination, namely, fluoroscopic study of the esophagus while the patient is swallowing a radiopaque mixture, i.e., barium sulfate or a bismuth compound. Under certain conditions barium-filled gelatin capsules are of value. No roentgen study of the esophagus should be considered complete until the chest has been studied fluoroscopically and fluoroscopic study of the esophageal function with a radiopaque substance has been made. With the present methods of roentgen study, which permit examination of the patient in various positions, it does not seem possible that the presence of esophageal disease can be overlooked. By these methods any alteration or irregularity in the size of the lumen can be detected, and the presence of strictures, sacculations or deviations from the normal can be ascertained. In most instances the roentgenologist's diagnosis based on a careful study of the esophagus and chest is correct. For absolute certainty it should, however, be supplemented by esophagoscopic data. Certain diseases of the esophagus may be confused with conditions involving the stomach or other portions of the gastrointestinal tract. This is more often observed with lesions involving the lower end of the esophagus. For this reason it is important to consider the esophagus as part of the alimentary tract and to include it in a routine roentgen study.

The esophagoscope provides a method of direct examination. It depends on objective evidence and should therefore be considered the final arbiter in diagnosis of esophageal disease. The interior of the entire esophagus may be minutely inspected; tissue, exudate and secretion may be removed and positive data secured. The esophagoscope is passed by sight. In the hands of one who has been trained in its use the procedure is safe. For this reason no mandrin or other device that would interfere with continuous vision through its lumen is used. As

the examination is done without general anesthesia, it may be repeated at weekly intervals or oftener if deemed necessary. As with most procedures that are noteworthy, special training is necessary to perform esophagoscopic examination with safety. The esophagoscope is not swallowed by the patient as is the stomach tube. The cricopharyngeus muscle is the stumbling block, and attempts by the inexperienced to pass the esophagoscope as they would a bougie will result in a large percentage of fatal perforations of the pharyngeal wall posterior to the cricopharyngeal fold.

Studies carried out in a haphazard manner usually result in the overlooking of certain examinations. A routine procedure should therefore be adhered to. The following diagnostic steps in order of procedure are suggested:

1. Complete history.
2. General physical examination, including the mouth, nose, throat, pharynx, larynx, pyriform sinuses and neck.
3. Serologic studies.
4. Fluoroscopic study of the chest.
5. Fluoroscopic study of the esophagus with an opaque mixture or a barium-filled capsule; taking of roentgenograms with the opaque mixture or the capsule or both.
6. Esophagoscopic examination, with a biopsy when indicated.

THERAPY

For a majority of esophageal diseases, treatment is either mechanical or surgical. In the presence of acute inflammatory conditions or of ulceration the esophagus should be placed at rest as far as possible by liquid diet or by gastrostomy. Of the medicaments, bismuth subnitrate is most commonly employed. It is given in doses of 5 to 15 grains (0.32 to 0.97 Gm.) dry on the tongue after eating and is swallowed without water. It exerts a protective action and is mildly antiseptic. For relief of pain, ethylaminobenzoate (anesthesin), 1 to 2 grains (0.06 to 0.13 Gm.), may be combined with the bismuth preparation. Alkalis are commonly employed in cases of peptic ulcer. Mild protein silver and silver nitrate are useful as topical applications. Accumulation of food in the esophagus proximal to a point of stenosis or in a diverticulum is relieved by swallowing water after eating in cases of slight retention or by esophageal lavage when the retention amounts to a considerable quantity. Dehydration in patients with esophageal stenosis is of serious consequence and should be combated by hypodermoclysis, proctoclysis and intravenous instillation. In cases of stenosis which cannot be promptly relieved, gastrostomy should be performed.

Congenital anomalies of the esophagus, such as atresia, usually terminate fatally within the first few days of life. Study of the esophagus with a small quantity of barium mixture is conclusive. Congenital webs or congenital stenosis with shortening of the esophagus and a thoracic stomach may go unrecognized for many years. The history is important. Patients with such conditions never have been able to swallow normally. In some cases superficial ulceration at the esophagogastric junction may develop. The symptoms simulate those of gastric ulcer, and the condition is often diagnosed as peptic ulcer of the stomach or of the esophagus. Roentgen study of the esophagus with the patient in the Manges "right lateral prone" position exhibits the characteristic dilatation of the stomach immediately above the diaphragm and stenosis at

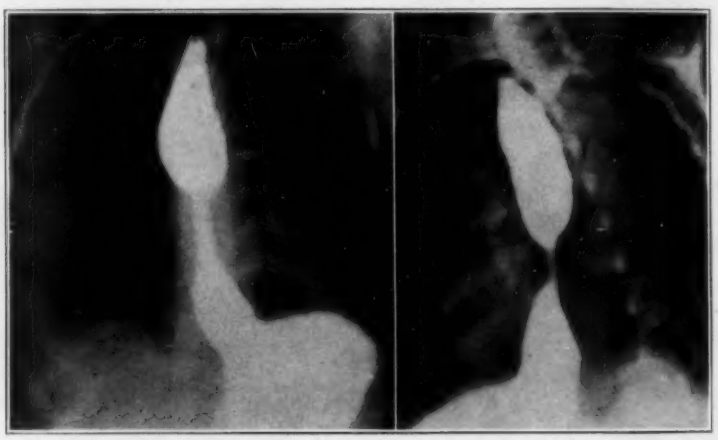


Fig. 1.—Roentgenograms of a boy aged 8 years who had had disturbances with swallowing since infancy. The esophagus was short and dilated; there was marked stenosis at the esophagogastric junction; a portion of the stomach was above the diaphragm, and there was absent the normal constriction of the food passageway at the level of the diaphragm. At esophagoscopic examination there was found ulceration at the esophagogastric junction (W. F. Manges).

the junction of the esophagus and the stomach (fig. 1). The esophagoscopic findings are conclusive.

Certain phases of the treatment are medical and include dietetic care, the employment of alkalis and posture. The stenosis may be dealt with esophagoscopically or by the use of an olive-tipped, fenestrated bougie passed over a previously swallowed thread. Topical applications of silver nitrate solution to the superficial areas of ulceration are helpful.

Deviation of the esophagus is observed in deformity of the vertebral column and is usually unassociated with symptoms. Deviation produced by aneurysm or mediastinal tumor is commonly associated with compression stenosis and is productive of disturbance in swallowing.

In the presence of acute esophagitis the substernal pain, dysphagia and occasional hematemesis may suggest some gastric disorder. There is commonly a history of trauma or of swallowing an irritant or a cauterant. Recent burns or scars about the mouth, throat and pharynx are significant. In the early stages, before healing and contraction of the ulcerated areas occur, the roentgen findings are usually inconclusive. It is important to recall that injury to the stomach may occur simultaneously if a cauterant has been swallowed, and the patient may complain of marked epigastric distress. If there remains a question as to the diagnosis, esophagoscopic examination may be performed. It would seem inadvisable, however, to do this immediately after injury, because of the danger of additional trauma and possible perforation of the esophagus.

Chronic esophagitis is commonly observed in persons with chronic alcoholism, in persons who habitually ingest highly seasoned, hot or irritating foods, in the presence of certain constitutional states and in cases of stenosis with retention of food and presents no problem in diagnosis if esophagoscopic examination is performed.

Benign ulceration of the esophagus, particularly in its lower third, may be confused with gastric disease. There may be dysphagia. Pain is a frequent symptom. It usually is referred substernally or to the region of the ensiform cartilage and may extend through to the back. Spastic stenosis involving either the cricopharyngeal muscle or the diaphragmatic pinchcock may occur. There is often associated esophagitis. Regurgitation and hematemesis may be observed. In cases of simple peptic ulcer of the esophagus, in those cases of congenital shortening of the esophagus and thoracic stomach in which there is ulceration at the esophagogastric junction and in cases of hiatal hernia, pain behind the lower end of the sternum extending to the back is significant. Odynophagia is common. Patients are conscious of food passing a tender point. Pain commonly begins immediately after eating and is promptly relieved by alkalis. Heartburn is a common symptom. This may be increased on the patient's assuming a prone posture. Dysphagia is usually not observed during the early stages. It may develop if stenosis occurs during healing. These ulcers commonly are superficial, covered with thin, grayish exudate and surrounded by a narrow inflammatory zone. They present no roentgen evidence unless there is secondary stenosis or deformity. The esophagoscopic appearances are characteristic; the diagnosis can be verified by histologic examination of the tissue.

Tuberculous ulceration of the esophagus is always associated with demonstrable tuberculosis elsewhere. If there is doubt, esophagoscopic examination, biopsy and bacteriologic studies will give conclusive findings.

Syphilitic ulceration may be associated with cancer or with secondary esophageal infection. A patient with carcinoma may have a positive Wassermann reaction. Biopsy should always be done. In questionable cases the therapeutic test is of value. Cancer is the most common cause of ulceration of the esophagus in adults. Roentgen study of the swallowing function and esophagoscopic examination followed by biopsy are conclusive diagnostic methods.

Vincent's infection, blastomycosis and the rarer lesions of the esophagus may cause confusion in diagnosis. The diagnosis is usually dependent on bacteriologic and histologic examination of material removed esophagoscopically.

Varicosities at the lower end of the esophagus may be the source of spontaneous hemorrhage. Varix practically never occurs in the upper part of the esophagus. If regurgitated, the blood is bright red and alkaline in reaction. More often, however, it is acid in reaction and of a "coffee ground" character, having been swallowed into the stomach and then vomited. Evidences of obstruction of the portal vein are important. The presence of varices at the lower end of the esophagus can be demonstrated esophagoscopically, but failure to observe them is not conclusive.

Stenosis of the esophagus may result from pressure due to periesophageal disease or from lesions in the esophageal wall itself.

Compression stenosis of the lower part of the esophagus may be produced by aneurysm, cardiac enlargement, enlargement of the left lobe of the liver, malignant disease of any of the intrathoracic viscera or lymphadenopathy. The common symptom is dysphagia. A general physical examination, serologic studies, roentgen study of the chest and functional studies of the esophagus will usually suffice to establish a correct diagnosis. If a question exists, esophagoscopic procedures may be used.

Cicatricial stenosis of the esophagus should not be confused with gastrointestinal disease, as the symptoms are practically always esophageal in origin, consisting of dysphagia, loss of weight (depending on the degree of stenosis) and regurgitation. Temporary lodgment of food producing complete obstruction, chronic esophagitis and occurrence of spasm, particularly if the stricture is at the lower end of the esophagus, may cause confusion in diagnosis. In addition, it must be recalled that patients who have swallowed strong acid solutions may sustain injury to the stomach as far as the pylorus. There may be a history of swallowing a corrosive substance, development of symptoms during one of the prolonged fevers, such as scarlet fever, peptic ulcer of the esophagus, syphilis or trauma following obstruction or prolonged sojourn of a foreign body. Roentgen study and use of the esophagoscope

should leave no doubt as to the diagnosis (fig. 2). The esophagoscopic findings of areas of scarring, granulation tissue with esophagitis and dilatation of the esophagus proximal to the point of stenosis are characteristic. It is of interest to note that in cases of esophageal stenosis of long standing the stomach is often contracted, probably from lack of distention by food and relative inactivity.

The treatment of strictures depends on the degree of narrowing and the extent of involvement. Dilation is a slow process and must be carried out over a long period. Interference with nutrition is a definite indication for gastrostomy. For a gastrostomized patient, the safest and best

Fig. 2.—Multiple strictures of the esophagus resulting from accidental swallowing of a concentrated solution of lye. There was moderate dilatation of the esophagus proximal to the strictures. Gastrostomy became necessary for feeding purposes. The strictures were dilated by retrograde bougienage (W. F. Manges).

method of treatment is retrograde bougienage. For patients not requiring gastrostomy, esophagoscopic bougienage or the passing of a fenestrated olive-tipped bougie over a previously swallowed string is the method of choice. Bougienage without a guide is hazardous and often results in perforation with fatal mediastinitis.

Spasmodic stenosis of the cricopharyngeus muscle is observed in cases of cancer of the lower end of the esophagus, of peptic ulcer and of diseases of the gastrointestinal tract. This condition may simulate and is often diagnosed as "globus hystericus." Such a diagnosis is justifiable only after every known diagnostic means has been utilized to rule out disease of the alimentary canal.

Cardiospasm, phrenospasm or preventriculosis, occasionally associated with disease of the stomach, biliary apparatus or appendix, is frequently mistaken for gastrointestinal disease. The common symptoms are discomfort or a sense of fulness referred to the epigastrium and relieved by regurgitation, inability to swallow certain foods, such as cold liquids, loss of weight (which may be marked) and, occasionally, severe pains about the region of the ensiform cartilage, extending through to the back. The cause is obscure. The occurrence of associated disease of the gastrointestinal tract or of the gallbladder is probably

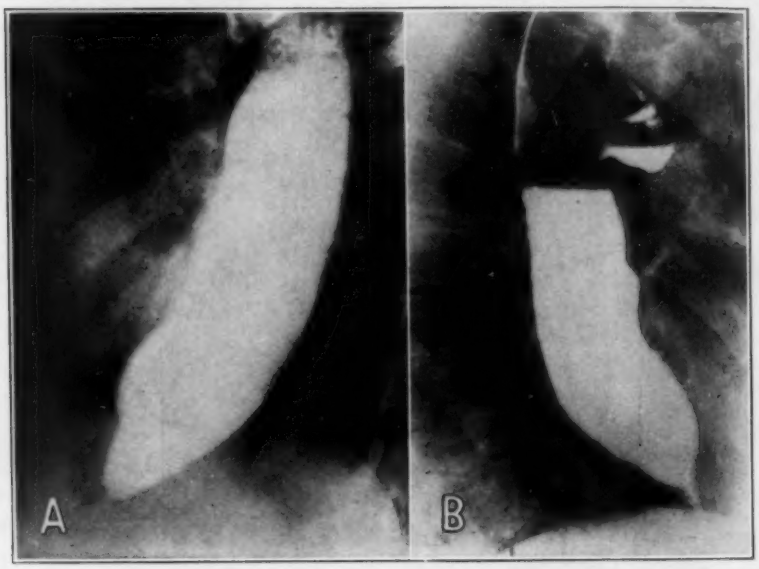


Fig. 3.—*A*, roentgenogram in a case of cardiospasm of seven years' duration occurring in a woman aged 32 years. The symptoms were dysphagia with liquids and solid foods, regurgitation of food and loss of weight. The esophagus was greatly dilated and terminated in a smooth, blunt end at the level of the diaphragm. At esophagoscopy there were found retention of food and chronic esophagitis. No difficulty was encountered in passing the esophagoscope through the hiatus esophageus into the stomach, demonstrating that there was an absence of organic stenosis. Prompt improvement followed aerostatic divulsion of the hiatus esophageus (J. T. Farrell Jr.). *B*, roentgenogram showing a traction diverticulum and a dilated esophagus suggesting cardiospasm in a man aged 56 years. Dysphagia, regurgitation and loss of weight were of six months' duration. At esophagoscopic examination there was found an organic stenosis of the esophagus at its lower end, apparently due to an infiltrating neoplasm. Abdominal exploration revealed an extensive carcinoma involving the cardia (M. Dillman).

coincidental. Deformity of the liver and endocrine disturbances have been described as etiologic factors. There is a dissociation of the swallowing mechanism, which probably is the result of disease of the

esophagus or of its nerve supply. Fibrosis of the esophageal wall has been a common finding. The degree of dilatation of the esophagus is variable. Dilatation is always present; in cases of extreme involvement the esophagus may hold as much as 2 liters. The regurgitated material is sour and stale and lacks all the constituents of gastric juice. Foods eaten twenty-four hours previously often can be identified. Unless the gastroenterologist is aware of this condition he will siphon off the test meal from the esophagus instead of from the stomach, and the analysis, lacking all constituents of gastric contents, will lead to erroneous conclusions. Patients with cardiospasm usually are unable to swallow a diagnostic tube. A roentgen check-up of the position of the tube should therefore precede an attempted gastric analysis in the case of a patient with cardiospasm or any condition associated with dysphagia. The diagnosis of cardiospasm can usually be made by the roentgenologist (fig. 3 *A*). The esophagoscope should always be used, however, to corroborate the diagnosis and to rule out cancer of the cardiac end of the stomach with the growth extending to the hiatus esophageus and producing esophageal symptoms simulating cardiospasm (fig. 3 *B*). The esophagoscopic appearances are characteristic. The thoracic portion of the esophagus is greatly dilated, and a large quantity of fluid and, at times, of residual food is present. The walls are thickened, at times leathery, and are thrown into numerous folds. The mucosa presents a furred or pasty appearance, suggesting a heavily coated tongue. Chronic esophagitis is present, and erosion of the mucosa may be observed. No resistance is encountered in traversing the hiatal orifice, although it often is difficult to find, owing to the numerous folds and the enormous dilatation. Antispasmodics are of value in occasional cases, although the condition is not primarily due to spasm. Regulation of the diet with avoidance of coarse foods, use of bismuth subnitrate dry on the tongue and esophageal lavage, particularly before retiring, are beneficial. None of these procedures, however, is entirely effective. The best results are secured by divulsion of the diaphragmatic pinchcock by hydrostatic or mechanical dilation. Relapses are not uncommon.

Diverticula of the esophagus are divided into pulsion and traction forms. The pulsion form is anatomically a diverticulum of the lower end of the pharynx (fig. 4, *A* and *B*). The neck or orifice of the pouch is at the level of the cricopharyngeus muscle, while the fundus extends downward behind the esophagus. It occurs more often in males than in females and is usually observed after the fiftieth year of life. A point of congenital weakness between the oblique and transverse fibers of the cricopharyngeus muscle is considered of etiologic importance. Cricopharyngeal spasm is unquestionably an added factor. Disturbance in swallowing is the first symptom noted. This may consist only of a gurgling sound when fluids are swallowed. Later there is actual dif-

ficulty in swallowing foods. As the pouch increases in size, it may exert sufficient pressure against the subdiverticular portion of the esophagus to produce marked dysphagia and seriously impair nutrition. Retention of food in the pouch results in gravitation of the food into the pharynx when the patient assumes a prone posture. This necessitates

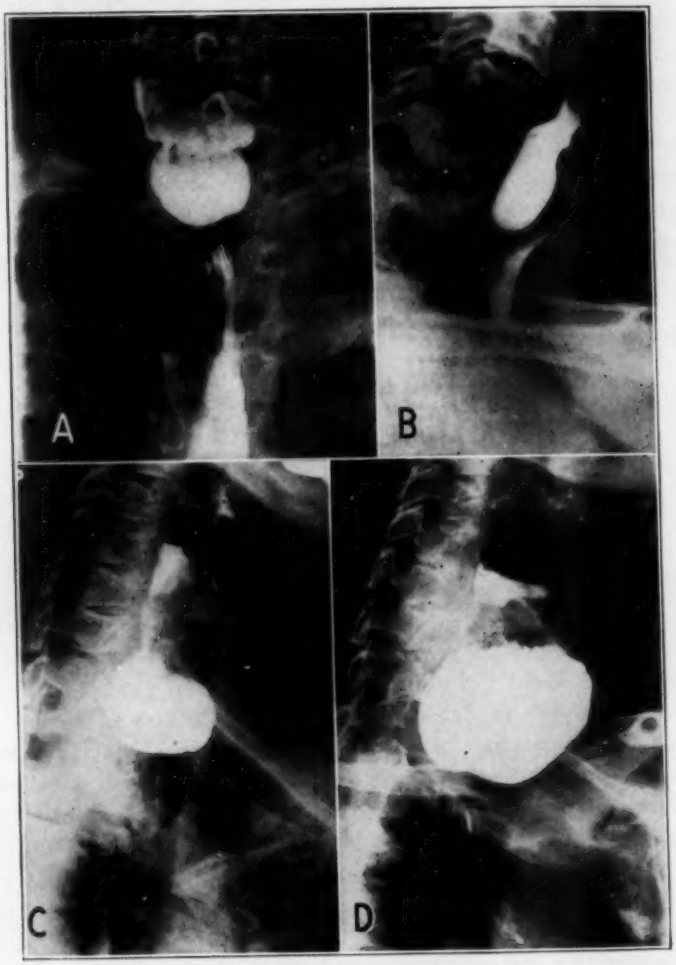


Fig. 4—*A* and *B*, roentgenograms of a pulsion diverticulum of the pharynx. In the lateral view, the partially filled pouch displaced the esophagus forward and narrowed the barium column, producing compression stenosis. The pouch was extirpated and the pharyngeal wall repaired by a one stage combined diverticulectomy (W. F. Manges). *C* and *D*, roentgenograms showing how the pouch in cases of pulsion diverticulum of the pharynx tends to increase progressively in size. Comparison of *C*, made in 1930, and *D*, made in 1934, showed a striking increase in the size of the pouch. There was marked dysphagia (J. T. Farrell).

frequent clearing of the throat and may cause choking sensations. The breath has a stale, sour odor. In cases of long-standing involvement, respiratory symptoms may result from aspiration of food particles and secretions, particularly while the patient is sleeping. Roentgen ray examination with a barium mixture gives practically conclusive results, and with esophagoscopic study the diagnosis becomes exact (fig. 4, *A* and *B*). Bougienage is inconclusive, unsafe and unnecessary. No organic stenosis of the esophagus is present. Dysphagia results from compression of the esophagus by the distended pouch. A diet of liquids and soft foods, lavage of the pouch immediately after eating and the use of bismuth subnitrate will allay the chronic inflammatory changes and prevent the distress caused by retained foods. Surgical extirpation of the pouch is the treatment of choice and should be done unless there is a contraindication. The pouch invariably becomes larger with increasing dysphagia, nutritional disturbances and respiratory symptoms from overflow of secretions (fig. 4, *C* and *D*).

The traction form of diverticulum occurs in the lower portion of the thoracic end of the esophagus and rarely causes symptoms. It is usually discovered accidentally by the roentgenologist in a routine gastrointestinal study. Traction diverticulum results from adhesions between the esophagus and surrounding structures, such as enlarged mediastinal lymph nodes or thickened pleura (fig. 3 *B*). Treatment is rarely required. In the presence of the occasional pouch with retention, posture may be advantageously employed.

Hiatal hernia, or hernation of a portion of stomach through the esophageal opening in the diaphragm into the thoracic cavity, may give rise to esophageal symptoms. There may be dysphagia, distress and even pain on swallowing foods, substernal pain, which often passes through to the back, and occasionally esophageal obstruction with regurgitation.

Results of studies made with the patient in the Manges or the Trendelenburg position are commonly characteristic (fig. 5). At esophagoscopic examination one finds gastric mucosa well above the level of the diaphragm; there is an absence of the normal hiatus or pinchcock appearance of the esophagogastric junction, and superficial ulceration may be observed at this point.

The treatment commonly is surgical.

Cancer of the upper end of the esophagus should not be confused with gastric disease. Difficulty in the localization of symptoms should always suggest that the entire esophagus must be considered when one is investigating esophageal or epigastric symptoms. The diagnosis of cancer commonly is made late, in spite of the fact that by careful roentgen study of the esophagus and by esophagoscopic examination one can recognize abnormalities that produce very moderate narrowing

of the esophageal lumen. Regurgitation, loss of weight, hematemesis and pain occur late in the disease and commonly represent a high degree of stenosis with periesophageal extension. Patients often will not consult a physician until the disease is advanced. Recognition of vague sensations on taking food, cricopharyngeal spasm or a feeling that food temporarily lodges in the esophagus in swallowing are symptoms of esophageal cancer, and prompt investigation of these by roentgen study and use of the esophagoscope, with biopsy, may aid in diagnosis early in the disease. Interpretation of these symptoms as evidences of a neurosis is inexcusable. The use of blind bougienage is unscientific and

Fig. 5.—Hiatal hernia of the stomach occurring in a woman aged 65 years. Dysphagia, "heartburn," and associated epigastric pain were of five years' duration. At esophagoscopic examination there was no stenosis of any part of the food passageway, and the normal hiatal constriction was absent. Superficial ulceration was present at the esophagogastric junction (K. Kornblum).

dangerous. Cancer involving the cardiac end of the stomach may first manifest itself by hiatal spasm; there may be extension upward, producing infiltrative stenosis (fig. 3*B*). Roentgen evidence of hesitation of a thick barium mixture at the hiatal level is an indication for direct inspection of the esophagus and stomach. The roentgen appearances of cancer are commonly diagnostic (fig. 6). Dilatation of the esophagus proximal to the growth is not so marked as that observed in cases of stenosis due to benign lesions.

Esophagoscopically, cancer of the esophagus is manifested by fungation, ulceration or infiltration. A specimen for biopsy may be safely taken from the areas of ulceration or fungation. In cases of compression stenosis or of submucosal infiltration with a normal overlying mucosa it is inadvisable to secure a specimen of tissue. Biting through a normal mucosa with tissue forceps opens avenues for infection.

Treatment of cancer of the esophagus has been extremely disappointing. Surgical extirpation should offer the greatest hope, but this is largely theoretic, as patients rarely are seen in the early stages of the disease and usually are poor risks for such a formidable procedure.

Fig. 6.—Irregular stenosis of the esophagus occurring in a man aged 52 years who complained of dysphagia and loss of weight. At esophagoscopic examination there was found an extensive fungating stenotic lesion beginning at the middle third of the esophagus. Biopsy was done. The histologic diagnosis was squamous cell carcinoma (K. Kornblum).

The results today from roentgen ray and radium treatment and from chemotherapy are not encouraging. The measures commonly employed are palliative, to prevent starvation. These consist of intubation of the esophagus, dilation of the cancerous stenosis and gastrostomy.

Foreign bodies in the esophagus should present no difficulties that might lead to confusion with gastrointestinal disease; it is a common experience, however, to see little children with esophageal foreign bodies who have been treated for gastric trouble because of regurgitation which

was erroneously interpreted as "vomiting." A roentgen study of the neck and chest often revealed a foreign body, esophagoscopic removal of which cured the patient of the "gastric trouble." Sharp, pointed or irregular objects lodged in the cervical portion of the esophagus may produce a sticking sensation or actual pain localized at the point of lodgment. In the thoracic portion of the esophagus localization is not so accurate. I observed a man who swallowed a denture which lodged in the esophagus and was localized roentgenologically at the level of the suprasternal notch. The symptoms were dysphagia and pain referred

Fig. 7.—Roentgenogram made after the patient had swallowed a barium-filled gelatin capsule, revealing lodgment of the capsule in the upper thoracic portion of the esophagus. There was a history of choking while eating fish, followed by a sticking sensation in the region of the suprasternal notch. At esophagoscopic examination a portion of fish bone was removed (W. F. Manges).

to a point 1 inch (2.5 cm.) below the ensiform cartilage, slightly to the left of the midline. After esophagoscopic removal of the denture the pain was promptly relieved. Foreign bodies, particularly food, may lodge in a stenotic esophagus narrowed by cicatricial changes, cancer or periesophageal disease.

In the case of foreign bodies that are opaque to the roentgen ray the diagnosis is obvious. It must be recalled that bones and other

nonmetallic materials, normally somewhat opaque to the roentgen ray, will not show on the film when superimposed on the shadows of the soft tissues and the bony skeleton. In these and in the nonopaque varieties, difficulty in diagnosis may be experienced; however, with the aid of a radiopaque material used in mixture or in capsule with the patient properly placed, the roentgenologist can give valuable data (fig. 7). In case of doubt, diagnostic esophagoscopic study is clearly indicated. The only method of treatment worthy of consideration is esophagoscopic removal.

"Retrograde esophagoscopy," the transgastric introduction of a retrograde gastroscope through a gastrostomy fistula, performed for purposes of alimentation, has greatly contributed to knowledge of the esophagus in the presence of stenotic or atresic lesions which could not be traversed by peroral esophagoscopic means. The technic is similar to that employed in retrograde gastroscopy. It is of great aid in placing an endless string in those cases of extensive cicatricial stenosis of the esophagus in which a string cannot be got through perorally for retrograde dilation. In these a flexible-tipped filiform bougie passed by retrograde esophagoscopic methods through the strictures can be recovered in the pharynx, a string secured to the bougie and the bougie with the string attached withdrawn through the esophagoscope. It is also indispensable in penetrating the cicatricial barrier by the combined peroral and retrograde method under fluoroscopic guidance in cases of cicatricial atresia of the esophagus.

1530 Locust Street.

CONGENITAL ATRESIA OF THE ESOPHAGUS

A STUDY OF THIRTY-TWO CASES

THOMAS H. LANMAN, M.D.
Assistant Professor of Surgery, Harvard Medical School; Visiting Surgeon, Children's Hospital
BOSTON

Congenital atresia of the esophagus is a rare anomaly. The experience of any one physician with this condition is necessarily limited. The literature on the subject is large, but most of the articles deal with a few individual cases plus a review of previously reported cases. The total number of reported cases is now about 300.

In the past eleven years, 32 cases of congenital atresia of the esophagus have been observed in the surgical service of the Children's Hospital. Thirty patients were submitted to operation, and complete autopsy was performed on the other 2 patients. In the 30 operative cases the surgical findings were supplemented by complete postmortem examination in 22 instances.

Cases observed in this hospital before 1929 are not considered here. Their inclusion would not change significantly the statistical data, and it is only since 1929 that a discussion of the forms of surgical treatment has been of value. Until then gastrostomy was the only surgical procedure used, and it was, of course, uniformly unsuccessful. This report is a critical analysis of 30 operative cases. All are from the surgical service of the Children's Hospital, and the operative procedures were carried out by a small group of closely associated surgeons, 16 of them being done by me.

There are many excellent reviews of reported cases, particularly those of Mackenzie,[1] Plass,[2] Hacker and Lotheissen,[3] Gage and Ochsner[4] and Rosenthal.[5] In many instances these reports include a

From the Surgical and Pathologic Services of the Children's Hospital and the Departments of Surgery and Pathology of the Harvard Medical School.

1. Mackenzie, M.: Malformations of the Esophagus, Arch. Laryng. **1**:301, 1880.

2. Plass, E. D.: Congenital Atresia of the Esophagus with Tracheoesophageal Fistula, Johns Hopkins Hosp. Rep. **18**:259, 1919.

3. Hacker, V., and Lotheissen, G.: Chirurgie der Speiserohre, in von Bruns, P.: Neue deutsche Chirurgie, Stuttgart, Ferdinand Enke, 1926, vol. 34, p. 192.

4. Gage, M., and Ochsner, A.: The Surgical Treatment of Congenital Tracheo-Esophageal Fistula in the New Born, Ann. Surg. **103**:725, 1936.

5. Rosenthal, A. H.: Congenital Atresia of the Esophagus with Tracheoesophageal Fistula: Report of Eight Cases, Arch. Path. **12**:756 (Nov.) 1931.

discussion of the embryologic factors causing this anomaly. Plass [2] and Rosenthal [5] in particular have given excellent descriptions of the embryologic considerations.

As this paper is primarily a discussion of the operative procedures, only a brief mention of the embryologic aspects of this anomaly is needed. Both the trachea and the esophagus are formed from the single tube of the primitive foregut. The lung bud appears as a projection on the anterior wall of the foregut at the region where the bifurcation of the trachea will occur, Normally the primitive foregut divides into two tubes, the trachea and bronchial tree anteriorly and the esophagus posteriorly. The division of this single tube into two occurs as a result of fusion in the midline of longitudinal projections arising from both lateral walls. The incomplete fusion of this septum at the level of the anteriorly growing lung bud results in the various types of anomaly observed. Strong and Cummins [6] have given an elaborate classification of all the various types and subdivisions of the anomalies that may occur.

Vogt [7] in 1929 published from this hospital an article in which he suggested for the variations of this anomaly a comparatively simple classification which meets all needs as far as clinical application is concerned. With type 1 there is a complete absence of the esophagus. This is extremely rare. With type 2 there is a blind end to both the upper and the lower segment of the esophagus, but there is no communication with the trachea from either segment. This is rare. With type 3 there are three varieties: With type 3a the upper segment communicates with the trachea, the lower segment being blind. This is rare. With type 3b the upper segment is blind, and the lower segment communicates with the trachea at or about the level of the bifurcation of the trachea. This is by far the commonest type. With type 3c both the upper and the lower segment have a communication with the trachea. This is rare.

In this series there were 19 boys and 13 girls. Twenty-nine, or 91 per cent, had anomalies of type 3b. One anomaly was of type 2, 1 of type 3a and 1 of type 3c. I have not seen any instance of type 1. These figures agree closely with those reported by others. Mackenzie [1] in 1880 reviewed 62 cases of esophageal atresia in which 40, or 60 per cent, of the patients showed a blind upper segment and a fistula between the lower segment and the trachea (type 3b). Hacker and Lotheissen [3] in 1926 published a series of 223 cases of which 173, or 77 per cent, had the same type of fistula of the lower segment. In 1931 Rosenthal [5] reported a series of 255 cases in which 205 patients, or 80 per cent, had

6. Strong, R. A., and Cummins, H.: Congenital Atresia of the Esophagus with Tracheo-Esophageal Fistula, Am. J. Dis. Child. **47**:1299 (June) 1934.

7. Vogt, E. C.: Congenital Esophageal Atresia, Am. J. Roentgenol. **22**:463, 1929.

this type of esophageal atresia with fistula. The more careful examination of such patients probably explains the more common finding of a tracheal fistula associated with the esophageal atresia. The associated tracheoesophageal fistula is the reason for many of the technical difficulties in operative treatment.

ASSOCIATED ANOMALIES

Many writers have called attention to the frequent association of other anomalies with tracheoesophageal fistula, particularly anomalies of the gastrointestinal tract. In this series there were 7 instances of associated anomalies of the gastrointestinal tract which if not treated by operation were in themselves incompatible with life. In 5 instances there was an imperforate anus, and there was 1 instance each of atresia of the ileum and atresia of the duodenum. One patient had both

Distribution of Various Types of Anomaly

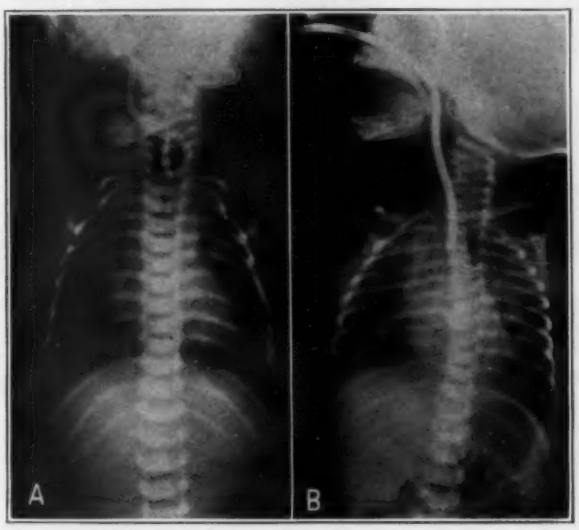
	Cases	Per Cent
Type 1	0	0
Type 2	1	3
Type 3a	1	3
Type 3b	29	91
Type 3c	1	3
	32	100

duodenal atresia and an imperforate anus, and another had a rectovesical fistula in addition to the imperforate anus. Other less important anomalies were Meckel's diverticulum, which occurred in 5 cases; various anomalies of the heart and blood vessels, which were found in 7 instances, and anomalies of the genitourinary tract, which were observed in 6 cases. The patients in case 8 and case 11 were children of the same parents.

SYMPTOMS AND DIAGNOSIS

The symptoms are characteristic and striking. They are attacks of choking and cyanosis occurring in the first few hours of life, and almost invariably these attacks are aggravated whenever any fluid is offered the infant. Attempts to swallow fluid are followed by immediate regurgitation and are usually accompanied with evidence of aspiration of fluid into the air passages, shown by choking, coughing and cyanosis. Between attacks it is usually noted that the infant has an excessive amount of mucus or saliva coming from the mouth. Often these secretions are described as frothy in appearance and indicate the overflow of secretions into the trachea. The aforementioned symptoms should suggest the diagnosis, and if the presence of an anomaly is suspected

the taking of a roentgenogram without the use of a contrast medium is helpful. Whether or not there is a fistula associated with the atresia, the giving of barium sulfate by mouth is accompanied by great danger and should never be done. Aspiration of barium sulfate alone into the air passages results in an irritative type of pneumonia. Aspiration of iodized poppyseed oil is relatively harmless in itself, but even with the most careful technic there will be some aspiration of the oil plus mucus and saliva containing the oral organisms from the upper segment of the esophagus. It is important also to realize that the use of a contrast medium will not give any helpful diagnostic information that cannot be obtained without its use. Before the plain roentgenogram is taken, a



A, roentgenogram taken without a contrast medium, showing the catheter obstructed in the upper segment of the esophagus. There is no gas in the stomach or bowel. This indicates that the anomaly is either type 2 or type 3a. *B*, roentgenogram taken without a contrast medium, showing the catheter obstructed in the upper segment of the esophagus and gas in the stomach and bowel. This is the commonest type of anomaly, type 3b. The rare type 3c would give a similar picture.

soft rubber catheter should be passed down the esophagus as far as it will go. The level of the upper pouch can be clearly demonstrated by this procedure. If there is no gas in the stomach or bowel, the anomaly is either type 2 or type 3a. If the catheter meets an obstruction and there is gas in the stomach and bowel, it is either type 3b or type 3c. For practical purposes this is all the differentiation needed preoperatively, as will be shown later (see accompanying illustration).

REPORT OF CASES

CASE 1.—J. F., a boy, entered the hospital Sept. 5, 1929, at the age of 12 days, with a typical history of attacks of choking, cyanosis and the immediate vomiting of fluids taken by mouth.

Roentgen examination with barium sulfate showed a blind upper segment of the esophagus; the lower end communicated with the trachea, as was shown by the gas in the stomach and bowel. The anomaly was of type 3b. There was evidence of bilateral pneumonic infiltration.

An anterior gastrostomy was done Sept. 5, 1929 (Dr. C. G. Mixter). The mediastinum was then explored through the back by the extrapleural approach. The lower segment of the esophagus communicated with the trachea just above the bifurcation. The blind upper end was somewhat dilated. The lower segment of the esophagus was separated from the trachea, and the fistula, which was about ⅛ inch (0.32 cm.) in diameter, was closed. The lower segment of the esophagus was freed; a catheter was inserted, and the opening of the lower part of the esophagus was sutured to the skin just to the right of the spine. The child died at the end of the operation. No autopsy was performed.

CASE 2.—M. McL., a girl, entered the hospital Dec. 29, 1929, at the age of 1 day, with a history of large amounts of mucoid saliva coming from the nose and mouth shortly after birth and also of "choking attacks."

Roentgen examination with barium sulfate showed atresia of the esophagus at the level of the bifurcation of the trachea. There was a considerable amount of opaque material in the main bronchial tree. The stomach and intestines contained a large amount of air, demonstrating a communication between the lower part of the esophagus and trachea. The anomaly was of type 3b. There was diffuse peribronchial congestion but no gross consolidation.

Operation (December 30) consisted of exploration of the mediastinum through the posterior extrapleural approach (Dr. W. E. Ladd). The upper segment of the esophagus was dilated and blind. The lower end of the esophagus joined the trachea just at the bifurcation. The communicating segment was doubly tied and cut between the ties. The lower segment of the esophagus was brought out through the back and sutured to the skin. The patient died just as the operation was completed.

Postmortem examination showed bilateral pneumonia and clearly demonstrated the danger of using barium sulfate in these cases. The bronchial tree in this case, as in the previous one, was filled with aspirated barium sulfate.

CASE 3.—J. C. P., a boy, entered the hospital July 10, 1930, at the age of 4 days, with a typical history of regurgitation of liquids associated with attacks of cyanosis and choking and of an excessive amount of "mucus" coming from the mouth and nose. Coarse rales were heard throughout both lungs, and roentgen examination showed the upper blind pouch of the esophagus at the level of the third dorsal vertebra and gas in the stomach and intestines.

Operation (July 10) consisted in exploration of the mediastinum through the posterior extrapleural approach (T. H. L.). The lower segment of the esophagus communicated with the trachea at its bifurcation. The anomaly was of type 3b. The communicating segment was doubly tied and cut between the ties. The lower segment of the esophagus was freed and sutured to the cutaneous wound. A better exposure was obtained in this case than in the 2 previous ones, but a small hole was made in the pleura. The child lived about twelve hours.

Postmortem examination showed bilateral pneumonia. There were bilateral megaloureters as an associated anomaly.

CASE 4.—J. P., a boy entered the hospital Aug. 17, 1930, at the age of 5 days, because of regurgitation of all fluid taken by mouth and because of bilateral pneumonia. Roentgen studies with barium sulfate had been done at another hospital. After transfusion the child improved slightly, and on August 18 an exploration of the mediastinum was made through the posterior extrapleural approach (Dr. G. D. Cutler). The upper segment of the esophagus ended blindly. The lower segment communicated with the trachea just above its bifurcation. The anomaly was of type 3b. The fistula was doubly tied and cut between the ties. The lower segment of the esophagus was brought out and sutured to the skin. The patient lived one hour.

Postmortem examination showed marked bilateral pneumonia and, as associated anomalies, Meckel's diverticulum and bilateral stenosis of the ureters with hydronephrosis.

CASE 5.—O. F., a boy, entered the hospital Oct. 29, 1930, at the age of 4 days, with a typical history of attacks of cyanosis, choking and an excessive amount of mucus in the mouth and larynx. Roentgen studies with barium sulfate had been made at another hospital, and atresia of the upper end of the esophagus had been demonstrated. The child was well developed but poorly nourished and in considerable respiratory difficulty. Coarse rales were heard throughout both lungs, and roentgenograms showed aspiration pneumonia and gas in the stomach and intestines.

On October 30 exploration of the mediastinum was made through the posterior extrapleural approach (Dr. C. G. Mixter). The upper segment of the esophagus ended blindly about 2 cm. above the bifurcation. The lower segment of the esophagus communicated with the trachea just above its bifurcation. The anomaly was of type 3b. The fistula was doubly tied and cut between the ties. The lower segment of the esophagus was freed, brought out and sutured to the skin. The patient lived fifty-six hours. Postmortem examination showed bilateral pneumonia and congenitally cystic kidneys.

CASE 6.—E. M., a boy, entered the hospital April 28, 1931, at the age of 4 days, with a history of regurgitation of all fluid taken by mouth. There had been no choking or cyanosis. The day before entrance he had several convulsions. He was a poorly nourished, dehydrated infant. Respirations were labored and difficult, and there were signs of bilateral pneumonia.

He improved slightly after a transfusion and parenteral fluids had been given, and on April 29 exploration of the mediastinum was done through a posterior extrapleural approach, but the patient died just as the mediastinum was exposed (Dr. G. D. Cutler).

Postmortem examination showed a tracheoesophageal fistula. The anomaly was of type 3b. There were also bilateral pneumonia, bilateral otitis media and meningitis.

CASE 7.—J. V., a boy, entered the hospital Sept. 3, 1932, at the age of 1 day, because of an imperforate anus.

As roentgen examination showed that the blind distal end of the sigmoid flexure was too high to reach from a perineal incision, a sigmoidostomy was done. The baby did well, but on the following day, on an attempt to pass a Levine tube, atresia of the esophagus was discovered. Roentgen studies with a catheter and

injection of iodized poppyseed oil showed that the upper segment of the esophagus ended in a blind pouch. There was a fistula between the lower segment of the esophagus and the trachea, as demonstrated by the gas in the bowel. The anomaly was of type 3b. The patient had an aspiration type of pneumonia and died on the third day. No postmortem examination was made.

CASE 8.—J. B., a boy, entered the hospital Dec. 2, 1932, at the age of 7 hours, because of an imperforate anus. As the blind end was too high to reach from a perineal incision, a sigmoidostomy was done on December 3. It was noted at the time of operation that the infant had considerable difficulty in breathing, and there were several attacks of cyanosis, which were relieved on suction of material from the mouth and throat.

In spite of the previous case, the possibility of tracheoesophageal fistula was not seriously considered until the following day, at which time roentgen studies showed complete obstruction of the upper segment of the esophagus and a large amount of gas in the stomach and intestines. On exposure (Dr. W. E. Ladd) of the posterior mediastinum a tracheoesophageal fistula (type 3b) was found, but the patient died on the table. No postmortem examination was made.

CASE 9.—R. H., a boy, entered the hospital Feb. 13, 1933, at the age of 3 days, with a typical history of regurgitation of all fluid given by mouth, at which times there were attacks of cyanosis. Roentgen studies with barium sulfate at another hospital had shown an obstruction of the upper segment of the esophagus. On admission the lungs showed some evidence of pneumonia, due in part at least to the barium sulfate, which had been aspirated into the lung.

On February 13 the mediastinum was explored through the posterior extrapleural approach (T. H. L.). The lower segment of the esophagus was identified and found to enter the trachea at its bifurcation. The anomaly was of type 3b. Two ties were placed about the fistula and cut between. The lower segment of the esophagus was freed; a soft rubber catheter was inserted, and the esophagus was brought out and sutured to the external wound.

The child did fairly well for nine days, but it was obvious that some effort must be made to exteriorize the upper end of the esophagus, because of the difficulty in preventing aspiration of the contents of the upper blind pouch. On February 25, through an anterior incision in the neck, the upper segment of the esophagus was freed, brought out through the neck and stitched to the wound. Its lower blind end was opened, and a small soft rubber catheter was inserted. This patient lived three days after the second operation and fifteen days after the first operation.

Postmortem examination showed bilateral pneumonia and some mediastinitis. In this case it was felt that a better exposure of the mediastinum was obtained by resecting the third, fourth, fifth and sixth ribs instead of the fourth, fifth, sixth and seventh ribs as in the previous instances. (See text for description of methods advised at present.)

CASE 10.—M. C., a girl, entered the hospital Aug. 13, 1933 at the age of 2 days, with a typical history of regurgitation of all liquid taken by mouth. There had also been several attacks of choking and cyanosis.

Roentgen studies with barium sulfate at another hospital demonstrated a blind upper segment of the esophagus, and there was barium sulfate throughout the entire bronchial tree. Roentgen examination here showed a large amount of gas in the stomach and intestines and some infiltration of the lungs. After parenteral fluids had been given, exploration of the mediastium through the posterior extrapleural

approach was done on August 13 (T. H. L.). The lower segment of the esophagus was found to communicate with the trachea at its bifurcation. The anomaly was of type 3b. The fistula was doubly tied and cut between the ties. A catheter was inserted in the lower segment of the esophagus. This lower segment was freed and brought out through the wound. The patient died twelve hours after the operation.

Postmortem examination showed bilateral pneumonia and bilateral pleurisy. In addition there were Meckel's diverticulum and an interventricular septal defect of the heart, and there was no gallbladder. Fluid from the chest grew Staphylococcus aureus. In this case there was a rather wide separation (3 cm.) between the upper and the lower segment of the esophagus.

Case 11.—M. L., a girl, entered the hospital Sept. 22, 1933, at the age of 15 days. As usual there was a history of an excessive amount of mucus coming from the mouth, and there had been attacks of choking and cyanosis whenever liquids were offered. Roentgen studies with barium sulfate at another hospital had demonstrated a blind end of the upper segment of the esophagus.

On September 23 the mediastinum was explored through a posterior extrapleural approach (T. H. L.). The third, fourth, fifth and sixth ribs were sectioned just to the right of the transverse processes, and about 1 inch (2.5 cm.) of each of these ribs was resected. The pleura was pushed laterally, and the mediastinum was entered. The lower segment of the esophagus communicated with the trachea at its bifurcation. The upper segment ended blindly. The anomaly was of type 3b. The fistula was doubly tied and cut between the ties. A soft rubber catheter was inserted in the lower segment of the esophagus which was freed, brought out through the wound and sutured to the skin.

The child stood the operation fairly well, and four days later (September 27) the upper segment of the esophagus was freed, brought out and sutured to the cutaneous wound of the neck. Its blind end was opened, and a catheter was inserted for drainage.

On September 30, seven days after the first operation, it was found that the opening of the lower segment of the esophagus had become detached from the cutaneous wound. It had retracted within the posterior thoracic wound, and it was no longer possible to introduce food through its opening.

On October 1 the posterior wound was opened, and the lower end of the esophagus was found and tied. It could not be brought out to the surface of the skin, as it had retracted and was adherent. It was felt that an attempt to free it would lead to further infection. A drain was replaced in the posterior wound, and an anterior gastrostomy was then done. For about two weeks after the anterior gastrostomy, the child did well and gained weight. She then had diarrhea; signs of bilateral pneumonia appeared; her course became downhill, and she died on October 23, at the age of 47 days. Death occurred twenty-five days after the third procedure and thirty-two days after the first operation.

Autopsy showed bilateral pneumonia, moderate mediastinitis, bilateral otitis media (Staph. aureus), bilateral hydronephrosis and stricture of the right ureter at the ureteropelvic junction.

Case 12.—M. B., a girl, entered the hospital Dec. 14, 1933, at the age of 2 days, with a history of attacks of cyanosis, dyspnea and choking. There was also an excessive amount of mucus coming from the mouth, which was relieved by suction. An attempt to pass a catheter showed an obstruction to the esophagus. Roentgenograms showed gas in the stomach and intestines and also considerable infiltration of both lungs and a probable aspiration type of pneumonia.

On December 14, exploration of the mediastinum was done through the posterior extrapleural approach (Dr. W. E. Ladd). The lower segment of the esophagus was found to communicate with the trachea. The anomaly was of type 3b. The fistula was doubly tied and cut between the ties. The lower segment of the esophagus was brought out through the wound; a catheter was inserted, and the esophagus was sutured to the skin. The patient died eight hours after the operation. No autopsy was performed. It is interesting to note that this patient's older brother had the same congenital anomaly (case 8).

CASE 13.—P. S., a girl entered the hospital Jan. 1, 1934, at the age of 11 days, on account of attacks of cyanosis, dyspnea and choking since birth whenever fluid was offered by mouth. The baby weighed only 3 pounds (1,307 Gm.), and there was roentgen evidence of bilateral pneumonia. A catheter met an obstruction in the esophagus at about the level of the second dorsal vertebra.

On January 2, exploration of the mediastinum through the posterior extrapleural approach was done (T. H. L.). The fourth rib was resected for about 2 inches (5 cm.), and the third and fifth ribs were cut close to the transverse process. The lower segment of the esophagus was found to communicate with the trachea at its bifurcation. The anomaly was of type 3b. There was better exposure of the site of the fistula than in any previous case. On account of the poor condition of the child, a double tie was placed about the fistula and cut between. It was planned to do an anterior gastrostomy later. However, the child died three hours after the operation. No autopsy was performed.

CASE 14.—J. T., a boy, entered the hospital Sept. 4, 1934, at the age of 2 days, with a typical history of attacks of choking, vomiting and cyanosis whenever fluid was given. Between attacks there was an excessive amount of mucus coming from the mouth. At another hospital a catheter had been passed, and complete obstruction of the esophagus had been demonstrated.

On admission scattered rales were heard throughout both lungs; the baby was poorly developed and poorly nourished and weighed only 5 pounds (2,268 Gm.). A catheter was passed down the esophagus, and an obstruction was met at the level of the first thoracic vertebra. There was no air demonstrable in the stomach or in the intestines. It was felt that the child had atresia of the esophagus, type 2; that is, both segments of the esophagus ended blindly. Two hours after admission the child had a severe attack of cyanosis, and respirations ceased.

Postmortem examination showed that the upper and lower segments of the esophagus were separated by about 5 cm., though there was a fine fibrous cord connecting the two. On careful dissection the upper segment of the esophagus was found to have a pin-sized fistula connecting it with the trachea. The anomaly, then, should be classified as type 3a and not type 2. There was marked bilateral pneumonia. Culture from the heart's blood and peritoneum showed Staphylococcus albus haemolyticus.

This mistake in diagnosing the type of anomaly is probably not of importance as regards surgical treatment. The fistula was so small that it is unlikely that there was any aspiration of material through it from the upper segment. No air had passed through it.

CASE 15.—J. C., a boy, entered the hospital on March 7, 1935, at the age of 1 day, with a typical history of cyanotic attacks occurring a few minutes after birth, which were relieved on suction of mucus from the mouth. These attacks continued, and on admission there was a very severe one. The lungs showed evidence of consolidation, and coarse rales were heard throughout both sides of the chest. Roentgenograms taken after passing a catheter showed the upper segment

of the esophagus to end blindly at about the level of the second dorsal vertebra, and there was considerable gas in the bowel. The child died a few hours after admission, no operation having been performed.

Postmortem examination showed a tracheoesophageal fistula of type 3b, bilateral bronchopneumonia of the aspiration type, atresia of the right ureter, Meckel's diverticulum and otitis media (Staph. aureus).

CASE 16.—M. C., a girl, entered the hospital Jan. 1, 1936, at the age of 2 days. There had been difficulty with breathing since birth and regurgitation of all fluid taken by mouth. On entrance there was some diminished resonance over the left side of the chest, and many coarse rales were heard in both lungs. A catheter passed down into the esophagus met an obstruction at the level of the second dorsal vertebra. There was considerable gas in the stomach and intestines.

On January 2, an exploration of the mediastinum through the posterior extrapleural approach was done (T. H. L.). About 1½ inches (3.7 cm.) of the fourth rib was resected, and the third and fifth ribs were sectioned close to the transverse processes. In pushing the pleura laterally a small hole was made in it. Good exposure was obtained. The upper segment ended blindly, and the lower segment of the esophagus was found to communicate with the trachea just above the bifurcation. The anomaly was of type 3b. The fistulous tract was doubly tied and cut between the ties. The upper and lower segments were then freed; both ends were opened, and an end to end anastomosis was performed with interrupted mattress sutures of fine silk. The wound was closed, a small rubber drain being left down to the mediastinum. The child's condition was good at the start but was rather poor at the end of the procedure, and he died three hours after the operation.

Postmortem examination showed bilateral acute bronchitis and a moderate amount of pulmonary atelectasis. The anastomosis appeared to be tight and not under undue tension.

CASE 17.—J. N., a boy, entered the hospital April 24, 1936, at the age of 4 days, because of persistent vomiting and regurgitation of everything taken by mouth and attacks of choking occasionally associated with cyanosis. Many coarse rales were heard throughout both lungs. Roentgen examination showed that the catheter passed down the esophagus met an obstruction at about the level of the second dorsal vertebra. There was a large amount of gas in the stomach and intestines, and there was also considerable infiltration in both lungs.

On April 25 the mediastinum was explored (Dr. W. E. Ladd) through the posterior extrapleural approach, and in this case 2 inches (5 cm.) each of the third, fourth, fifth and sixth ribs was removed. The lower segment of the esophagus was found to communicate with the trachea at the bifurcation. The upper end was blind. The anomaly was of type 3b. The fistula was ligated and cut; the lower segment of the esophagus was freed and brought out through the wound after a catheter had been placed in the esophagus.

The patient died two hours after operation. No autopsy was performed.

CASE 18.—C. L., a boy, entered the hospital June 14, 1936, at 8 days of age, because of regurgitation associated with attacks of cyanosis and choking on attempted feeding. Roentgen studies with barium sulfate at an outside hospital showed atresia of the upper segment of the esophagus. On admission roentgen examination showed considerable infiltration of both lungs and evidence of aspiration pneumonia; there was a considerable amount of barium sulfate in the bronchial tree. A large amount of gas was seen in the stomach and intestines, and it was presumed that the anomaly was of type 3b.

On June 14, the mediastinum was explored through a posterior extrapleural approach, with resection of a portion of the fourth and fifth ribs, the third and sixth ribs being sectioned close to the transverse processes (T. H. L.). Good exposure of the mediastinum was obtained. The upper segment of the esophagus was not seen. The lower segment of the esophagus communicated with the trachea at the bifurcation. The fistulous tract was doubly tied and cut between the ties. After a catheter had been placed in the lower segment of the esophagus, it was freed and brought out through the wound. The child did fairly well, but, on account of the constant aspiration of mucus from the upper segment, on June 25, ten days later, the upper segment of the esophagus was dissected as far as possible, doubly tied, cut between the ties and sutured to the skin on the anterior surface of the neck. In this case complete dissection of the upper segment was not accomplished. On June 29, it was found that the lower segment of the esophagus had separated from the cutaneous wound and had retracted within the chest.

On June 30, an anterior gastrostomy was done by the Witzel technic. The sutures and drain were removed from the anterior upper wound, and there was no evidence of infection in the neck. There was a considerable amount of saliva draining from the upper esophagostomy opening. At this same time the posterior esophagostomy wound was explored. The tissues were very friable, and there was evidence of considerable infection in this area. The open end of the lower segment of the esophagus was not seen. This posterior wound was left open with a drain. The child did fairly well for a week, after which he began to go downhill. He died on July 12.

Postmortem examination showed a rather marked degree of mediastinitis, bilateral pneumonia with multiple small pulmonary abscesses, moderate fibrinous pleuritis on the right, otitis media and peritonitis. Culture of the exudate in the right pleural cavity grew gram-negative cocci and gram-negative bacilli.

This attack on the problem brings out a number of points. This case, as well as case 11, shows that if the patient lives it is difficult to keep the exteriorized lower end of the esophagus attached to the external wound in the back. Unless the lower segment is freed for a considerable distance, the tension will cause it to retract. If it is sufficiently freed, the blood supply at its open end is probably interfered with. Also it seems clear that unless the upper segment is exteriorized the patient will continue to be in danger of having an aspiration type of pneumonia. In this instance postmortem examination showed that there had been a small fistula connecting the distal stump of the upper segment of the esophagus with the trachea. This must have been an added factor in the aspiration of the contents from the upper segment. The anomaly in this case should be classified as type 3c.

Case 19.—J. T., a boy entered the hospital July 11, 1936, at the age of 2 days. All fluids given by mouth were immediately regurgitated or vomited, and there was an excessive amount of mucus coming from the mouth. There had been occasional attacks of cyanosis. On examination the child appeared in fairly good condition, though coarse rales were heard throughout both sides of the chest. Roentgen examination showed complete obstruction of the upper segment of the esophagus and considerable gas in the stomach and intestines. The anomaly was of type 3b.

On July 11, about 2 inches (5 cm.) of the fourth rib was resected (T. H. L.), and the third and fifth ribs were sectioned close to the transverse process. The intercostal muscles were separated, the pleura pushed laterally and the mediastinum entered. A small rent was made in the pleura, but this was resutured. The lower segment of the esophagus was found to communicate with the trachea at the bifurcation. The fistulous tract was doubly tied and cut between the ties, and

the lower segment was freed. The upper end was identified and freed. The two ends were cut and anastomosed with interrupted mattress sutures of silk.

The patient stood the operation fairly well. The exposure was good, and it was felt that the anastomosis had been done without tension. During the following forty-eight hours the infant's respirations became very labored, and he died seventy-two hours after the operation. No postmortem examination was made.

Case 20.—J. O. B., a boy, entered the hospital Nov. 30, 1936, at the age of 4 days. There was a typical history of difficult breathing and attacks of cyanosis whenever liquid was offered by mouth. Examination showed a moderate degree of icterus; there were frequent mild attacks of labored breathing associated with cyanosis, and there was an excess of mucus coming from the mouth. Rales were heard throughout both lungs and some harsh breath sounds over the right lung. Roentgenograms showed that the catheter met an obstruction at the lower level of the second dorsal vertebra and that there was a large amount of gas in the stomach and intestines. The anomaly was of type 3b.

Operation was performed November 30 (T. H. L.) with the same approach as in the previous case. The lower segment of the esophagus was found to communicate with the trachea through an unusually wide fistula. The fistula was doubly tied and cut between the ties. The lower segment was mobilized and opened; the upper blind segment was also freed and opened. An anastomosis was done with several mattress sutures of fine silk, and there appeared to be no tension on the suture line. A small rent had been made in the pleura, but this was closed. A rubber drain was left in the mediastinum, and the wound was closed.

This patient lived eight days and at the end of this time, perhaps unwisely, the patient was given a small amount of sterile saline solution by mouth, which was swallowed easily. The following day a slight serous drainage was noted coming from the wound. This was thought to contain saliva. The same day the baby's condition suddenly became poor; there was circulatory collapse, and he died on the ninth postoperative day.

Postmortem examination in this case was rather discouraging in that it showed a leak at the site of the anastomosis. It was felt that this was due to tension at the site of the anastomosis rather than a direct result of the fluid given by mouth. There was acute posterior mediastinitis; Streptococcus haemolyticus was isolated. There was a slight amount of fluid in both lungs, which on culture grew Str. haemolyticus. There was bilateral pneumonia. Str. haemolyticus was isolated from the blood stream. Meckel's diverticulum, a hemivertebra and an anomaly of the pancreatic duct were associated with the condition.

In spite of this fatality, it was felt that progress in treating this condition by the direct attack was being made. At least there was no undue operative shock, as the patient lived over a week.

Case 21.—M. O'S., a girl, entered the hospital May 12, 1937, at the age of 6 days, because of attacks of vomiting, regurgitation of fluid and cyanosis since birth. There was a moderate amount of abdominal distention, and there was an ectopic anus with a narrow opening at the junction of the posterior wall of the vagina and the perineum. Both lungs showed evidence of a pneumonic process.

In spite of the poor condition of the child, an attempt was made after giving a transfusion and parenteral fluids to explore the posterior mediastinum through the extrapleural approach (T. H. L.). When the mediastinum was entered it was found that the lower segment of the esophagus entered the trachea at a lower level than usual, appearing as a direct continuation of the trachea. The upper segment was found to end blindly; there was a rather wide separation (3 cm.)

between the upper and the lower segment of the esophagus. The patient's breathing ceased before any further operative procedures were carried out.

Postmortem examination showed, in addition to the tracheoesophageal fistula (type 3b), an ectopic anus, infantile coarctation of the aorta, anomalous and rudimentary attachment of the mesentery of the bowel, acute bilateral bronchopneumonia, aspiration of amniotic sac contents and acute mastoiditis on the left. Had this patient lived an attempt would have been made only to close the fistula at that time. There was too wide a separation between the two segments to justify trying to anastomose them. Additional later procedures would have been necessary.

Case 22.—J. G., a boy, entered the hospital July 26, 1937, at the age of 2 days. There had been regurgitation with some cyanosis on attempted feeding. Examination under roentgen rays at another hospital had not included the use of opaque fluid. The child was in fairly good condition. Roentgen examination at this hospital showed that the catheter met an obstruction at the fourth thoracic vertebra and that there was gas in the stomach and in the upper part of the intestinal tract. The anomaly was of type 3b.

On July 27, the mediastinum was explored through the posterior extrapleural approach (T. H. L.). About 1 inch (2.5 cm.) of each of the fourth and fifth ribs was resected, and the third and sixth ribs were sectioned close to the transverse process. This gave an excellent exposure, and the lower segment of the esophagus was found to communicate with the trachea at its bifurcation. The fistulous tract was doubly tied and cut between the ties. The lower and upper segments were then freed, and it was possible to bring them together without tension. The upper segment of the esophagus was opened at its lowest point, and the two ends of the esophagus were then anastomosed with interrupted mattress sutures of fine silk. A rubber drain was placed in the mediastinum, and the wound was closed.

The patient recovered from the operation and with administration of parenteral fluids his condition greatly improved. He was given nothing by mouth. There was a slight serous discharge from the posterior wound on the fifth day. Parenteral fluids were given daily, as well as several blood serum tranfusions. On the ninth postoperative day the respirations suddenly became labored, and the child died in a very few minutes.

Postmortem examination showed that the two ends of the esophagus were approximated, and, although there was slight tension in this region, there was no evidence of leakage. The wound appeared to be healing well, and there was little, if any, mediastinitis. Cultures showed no growth. It was felt that the anastomosis had been adequately done. There were dilatation of the right side of the heart, hemorrhage in the lungs and free fluid in the serous cavities. There seemed little doubt that death was caused by too much fluid given parenterally. A constant intravenous infusion was used, and this I now realize to be very dangerous for patients of this age group. Distressing as was the result in this case, it shows that a direct attack with primary anastomosis may be successful.

Case 23.—F. R., a girl entered the hospital Aug. 26, 1937, at the age of 36 hours, because of an imperforate anus. Operation to correct this was done on August 27. It was noted at the time of operation that the baby had attacks of cyanosis and that there did not seem to be as much meconium in the rectum as is usual. On the second day moist rales were heard in the chest, and there were some attacks of cyanosis. Roentgen examination showed that the catheter had met an obstruction at the level of the fourth dorsal vertebra. There was a considerable amount of air in the stomach and intestines.

On August 28, the posterior mediastinum was explored through the extrapleural approach (Dr. G. D. Cutler). The upper segment of the esophagus was found to end blindly; while an attempt was made to identify the lower end the patient's condition became very poor, and he died on the table.

Postmortem examination showed a tracheoesophageal fistula type 3b. There were also Meckel's diverticulum, marked bilateral bronchopneumonia and the postoperative repair of an imperforate anus. In view of cases 7 and 8 in which there was an imperforate anus in addition to a tracheoesophageal fistula, this condition might have been recognized sooner.

CASE 24.—J. G., a boy, entered the hospital Oct. 1, 1937, at the age of 48 hours, with a history of repeated attacks of cyanosis and choking whenever liquid was offered. There was also a note that nothing had been passed by rectum.

On examination coarse rales were heard throughout both lungs. Roentgen examination showed that the catheter met an obstruction at the level of the first dorsal vertebra. There was no gas in the stomach or intestines. A diagnosis of esophageal atresia, type 2 (see illustration, *B*) was made, and it was felt that this type of anomaly in the presence of pneumonia would be better attacked by an anterior esophagostomy of the upper, blind end followed by an anterior gastrostomy. This was done on October 1 (Dr. P. J. Mahoney). The child did fairly well at first, but on the seventh day his condition became poor; there was evidence of bilateral pneumonia, and he died on the eighth postoperative day.

Postmortem examination showed esophageal atresia of type 2; that is, both ends were blind, neither segment communicating with the trachea, and the two blind ends were separated by about 5 cm. There were bilateral pneumonia, dextraposition of the aorta and an interventricular septal defect. There was an area of erosion in the stomach, with perforation and peritonitis. This area was not associated with the gastrostomy wound, and its cause was obscure. Microscopically the wall of the stomach showed an extensive infiltrative reaction with autolysis but without evidence of inflammatory reaction. The observation in this case of cerebral cortical atrophy raises the question whether this gastromalacia might be similar to the Cushing ulcers associated with lesions in the region of the third ventricle. In only 1 other case has there been observed such a wide separation of the two segments of the esophagus.

CASE 25.—M. W., a girl entered the hospital July 24, 1938, at the age of 3 days, with a history of difficulty in swallowing and occasional attacks of choking. On admission the child was in rather poor condition. Roentgen examination with a catheter passed down the esophagus showed that the upper end of the esophagus ended blindly and that there was a large amount of gas in the stomach and intestines. There was pneumonic infiltration in both lung fields. Operation, performed on July 24 (Dr. P. J. Mahoney), consisted in exploration of the mediastinum by the transpleural approach. The patient died just as the mediastinum was entered. Postmortem examination showed marked pneumonia on both sides and a tracheoesophageal fistula of type 3b. It appeared as though the two segments were close enough to permit anastomosis. In addition there were dextraposition of the descending thoracic aorta, a slight degree of infantile coarctation of the aorta and a patent ductus arteriosus. Death was due to surgical shock as well as to a bilateral bronchopneumonia. This was the first attempt in this hospital of a transpleural approach to the mediastinum.

CASE 26.—M. H., a girl, entered the hospital Dec. 26, 1938, at the age of 1 day, because of an imperforate anus and occasional attacks of cyanosis. Roentgen examination showed a pneumonic process in both lungs. A catheter was not

passed down the esophagus. There was a collection of gas in the upper portion of the abdomen, which gave the appearance of obstruction of the duodenum with dilatation of the stomach and duodenum. There was no gas in the intestines distal to the duodenum. No estimate could be made of the extent of the intestinal atresia.

On December 27, an abdominal exploration was made (T. H. L.). Duodenal atresia was found, which was relieved by a side to side duodenojejunostomy. The remainder of the intestinal tract seemed patent. On the next day, operation for imperforate anus was undertaken. The rectum was easily freed, opened and sutured to the edge of skin without tension. The child died within twelve hours after the second operation. Postmortem examination showed, in addition to the findings already known, a tracheoesophageal fistula of type 3b. There was extensive bilateral bronchopneumonia. There were an interventricular septal defect of the heart and almost complete stenosis of the right ureter. The two operative procedures for the two recognized lesions seemed adequate.

Case 27.—J. F., a girl, entered the hospital on March 15, 1939, at the age of 3 hours, on account of an imperforate anus. Attacks of slight cyanosis had been noted soon after birth. On admission the respirations were labored, but nothing abnormal was noted in the chest. There was no anal opening. There appeared to be a bifid scrotum, above which was a rudimentary structure thought to be a penis or possibly the clitoris. No testes were felt. On account of increasing cyanosis an attempt was made to suction fluid from the mouth, but it was found that a catheter could not be passed into the stomach. There was much gas in the stomach and intestines. In view of the experience in other cases of tracheoesophageal fistula associated with an imperforate anus, it seemed rational to attack the fistula first, as the more urgent anomaly.

On March 15, the mediastinum was exposed (T. H. L.) through a transpleural approach between the fourth and fifth ribs. The posterior mediastinum was entered. The lower segment of the esophagus was found to enter the trachea just above its bifurcation, and the blind upper segment was found 1 cm. above it. Both ends were freed, and an anastomosis was performed with interrupted mattress sutures of silk, but before this operation could be completed the patient died.

Postmortem examination showed that the anastomosis was well performed and not under tension, but, of course, whether it would have remained tight cannot be stated. In addition there was the known atresia of the rectum; there were also a bicornuate uterus, a patent ductus arteriosus, a persistent cloaca, a cystic left kidney and hyperplasia of the clitoris. In this case, as in case 27, there seemed to be considerable shock associated with the transpleural approach.

Case 28.—J. K., a boy, entered the hospital May 20, 1939, at the age of 2 days, with a typical history of regurgitation of large amounts of mucus, attacks of cyanosis and choking spells associated with the giving of fluid by mouth. There was also marked abdominal distention, and nothing had been passed by rectum. A diagnosis of intestinal obstruction in addition to a probable tracheoesophageal fistula was made.

Abdominal exploration (T. H. L.) showed that the small intestine was dilated and gangrenous from about 8 inches (20.3 cm.) below the ligament of Treitz to just distal to the ileocecal valve. The bowel was obviously not viable. Resection and anastomosis between the remaining small intestines and the ascending colon were done. In view of the tracheoesophageal fistula, the cardiac end of the stomach was then ligated, and a gastrostomy was performed below this ligation. In spite of these heroic measures, the justification for which is debatable, the patient did not die until the third postoperative day.

The postmortem observations, in addition to the conditions noted at operation, were a tracheoesophageal fistula of type 3b, bilateral pneumonia, bilateral pleuritis, some pulmonary edema, cerebral congestion, coarctation of the aorta, stenosis of the right ureter and bifid pelvis of the left kidney. The cardiac end of the stomach was well closed. There was some free fluid in the peritoneal cavity, but there was no peritonitis.

CASE 29.—Y. O., a girl, entered the hospital Aug. 20, 1939, at the age of 6 hours, because of repeated attacks of cyanosis. The child weighed 3 pounds 8 ounces (1,533 Gm.) and was in poor condition. At entrance it was felt that some intracranial injury might explain the symptoms, and it was not until the third day that a tracheoesophageal fistula was suspected and demonstrated. There had not been any attack of vomiting, and, although the fluid given by mouth was regurgitated, it was felt that this was merely an evidence of inability to suck.

Roentgen examination showed obstruction in the esophagus with air in the stomach. The anomaly was of type 3b. On account of the poor condition of the child it was decided to attempt the procedure advocated by Gage and Ochsner,[4] as in case 28. Through an abdominal incision the cardiac end of the stomach was tied, and a small catheter was sewed in the fundus of the stomach. The child's condition remained poor, and she died twenty-four hours after the operation. No autopsy was performed.

CASE 30.—W. M., a boy, entered the hospital Aug. 20, 1939, at the age of 3 days, with the history that there had been an excessive amount of mucus coming from the mouth, with vomiting and attacks of cyanosis and choking on attempts at feeding. Coarse rales were heard throughout both sides of the chest. Roentgen examination showed that the catheter met an obstruction at the third dorsal vertebra, and there was considerable gas in the stomach and intestines.

Operation was done on August 21 (T. H. L.). Through an abdominal approach the Gage-Ochsner procedure was used. The cardiac end of the stomach was tied, and a gastrostomy of the Witzel type was performed. After the operation the patient continued to have cyanotic spells in spite of constant efforts to keep the upper segment of the esophagus free of secretions. These increased in severity, and on the third postoperative day the child died during an attack.

At autopsy there were bilateral pneumonia and peritonitis probably due to leakage above the gastric tube. The tracheoesophageal fistula was of type 3b.

CASE 31.—C. R., a girl, entered the hospital Jan. 16, 1940, at the age of 3 days. There was the usual history of attacks of cyanosis and vomiting immediately after feedings were attempted. She appeared to be a mongol, weighed only 4 pounds (1,814 Gm.) and was in poor condition. There was evidence of involvement of both lungs. A catheter passed down the esophagus met an obstruction at the level of the second dorsal vertebra, and there was considerable gas in the stomach and intestines. It was decided to attempt the procedure suggested by Gamble.[8] The abdomen was opened (T. H. L.), and the stomach was cut entirely across at the junction of the upper and middle third. The upper end was sutured to the upper end of the wound to allow free drainage of the lower segment of the esophagus. The lower part of the stomach was closed, and a catheter was introduced in this lower segment by the Witzel technic. The patient lived only eighteen hours.

Autopsy showed bilateral pneumonia, bilateral pleurisy, a patent foramen ovale and a horseshoe kidney with three ureters. There was no peritonitis, and the

8. Gamble, H. A.: Tracheo-Esophageal Fistula, Ann. Surg. **107**:701, 1938.

gastrostomy openings seemed tight. However, it was too soon after operation for this to be significant.

CASE 32.—H. G., a boy, entered the hospital Feb. 8, 1940, at the age of 1 day. There had been a large amount of mucus coming from the mouth, but there had been no cyanotic attacks. It was noted that the child had an imperforate anus, and there was a history of meconium being passed from the bladder. Roentgen examination showed that a catheter passed down the esophagus stopped at the level of the second dorsal vertebra. There was gas in the stomach and bowel. A film taken in the upside-down position showed that the gas in the blind end of the sigmoid flexure was at the brim of the pelvis.

Operation was performed on February 8 (T. H. L.). A tube was placed in the lower blind end of the sigmoid flexure. The large bowel was greatly dilated with meconium; some areas of the bowel wall were very dark, but it seemed viable. The sigmoid flexure was sutured to the peritoneum about the tube, and the wound was closed. An upper left rectus incision was then made, and a very small stomach was delivered into the wound. It was cut across at the junction of the upper and middle third as suggested by Gamble.[8] The distal end of the stomach was then closed, and a catheter was inserted and sutured in the distal segment for feeding. Instead of attempting to suture the opened cardiac end of the stomach to the wound, the cardia was closed except for about ½ inch (1.2 cm.) on the greater curvature, where a catheter was inserted up into the esophagus and sutured in place. This step is a modification of both the Gamble[8] and the Leven[9] procedure. The upper gastrostomy tube was sutured to the upper end of the wound; the peritoneum was brought together in the middle of the wound, and the lower gastrostomy tube was sutured to the lower end of the wound. Muscle, fascia and skin were sutured between the two catheters. The liver interfered considerably with the delivery of the stomach in this case. The child died forty-eight hours after the operation, and postmortem examination showed that the tubes were not leaking but that there was some blood in the peritoneal cavity, and there appeared to be early peritonitis. There were bilateral pneumonia and collapse of the upper lobe of the right lung. The tracheoesophageal fistula was of type 3b.

COMMENT

Although Richter[10] in 1913 advocated a direct attack on the posterior mediastinum, with exposure of the esophagus and trachea, this was considered a procedure of too great magnitude for an infant, and even today there are many who feel that this is so.

In 1929 there was the first attempt at this hospital of any procedure other than a gastrostomy. In case 1, Dr. C. G. Mixter exposed the mediastinum through a posterior extrapleural approach. This approach had been successful in draining the mediastinum in a previous case of purulent mediastinitis caused by a foreign body perforating the esophagus. In cases 1 and 2 the attempt was successful in closing the fistula and in bringing the lower segment of the esophagus out through the back to

9. Leven, N. L.: Surgical Management of Congenital Atresia of the Esophagus with Tracheo-Esophageal Fistula, J. Thoracic Surg. **6**:30, 1936.

10. Richter, H. M.: Congenital Atresia of the Esophagus: An Operation Suggested for Its Cure, Surg., Gynec. & Obst. **17**:397, 1913.

be used for feeding. In both instances, however, the patient died almost immediately after the operation. In case 3 the same procedure was used, and the child lived for twelve hours, and in case 5 the patient survived for thirty-six hours.

In 1933 the posterior approach was used in case 9; the fistula was closed and the lower segment of the esophagus exteriorized. The infant survived nine days, at the end of which time the upper segment of the esophagus was exteriorized through an anterior incision in the neck. This patient lived fifteen days after the first operation and three days after the second.

In case 11 the posterior approach to the mediastinum was made; the fistula was tied; the lower segment of the esophagus was exteriorized to the back, and four days later the upper segment of the esophagus was exteriorized through an anterior incision in the neck. Seven days later an anterior gastrostomy was done, and at the same time the posterior esophagostomy was closed. The patient lived twenty-five days after the third operative procedure and thirty-two days after the first operation. This case offered some encouragement, although it was realized that had the child survived there would still have been the problem of connecting the openings of the "upper esophagostomy" with the anterior gastrostomy.

With increasing experience a better and better exposure of the posterior mediastinum was obtained, and it was possible to do this without making extensive tears in the pleura. The operative procedure then consisted in subperiosteal resection of the third and fourth ribs for a distance of about 1½ to 2 inches (3.7 to 5 cm.), and section of the ribs above and below close to the transverse processes, the pleura being pushed laterally and the mediastinum entered.

Case 16 (1936) represents the first attempt at direct anastomosis through the extrapleural approach of the upper and lower segments after the fistula had been tied. This was successfully done, but the patient died a few hours after the operation. In case 18 a posterior esophagostomy of the lower segment after closure of the fistula was done. One week later the upper segment was exteriorized through an anterior incision in the neck. Four days later an anterior gastrostomy was done as in case 11. The lower segment of the esophagus had separated from the posterior cutaneous wound and had retracted within the chest. This patient died twenty-eight days after the first operation and thirty-six days after birth. (See report of case 18).

In case 19, direct anastomosis after tying the fistula was tried again. In this instance the patient lived seventy-two hours after operation. In case 20 the same procedure was used, and the patient lived eight days. (See report of case 20.)

The operation in case 22 (1937) was the most nearly successful of all. A posterior extrapleural approach was used; the fistula was closed,

and an anastomosis of the upper and lower segments was performed. The infant lived nine days, and postmortem examination suggested that death was due solely to overhydration with parenteral fluid. (See report of case 22.)

The extrapleural approach required a good deal of time, so that in case 25 a transpleural approach to the mediastinum was used as practiced by Churchill.[11] The patient, however, was not in good condition and died of surgical shock on the table. The condition in case 26 was hopeless, as duodenal atresia and an imperforate anus were also present. In case 27 there was an imperforate anus as well as atresia of the esophagus. In case 28 there was atresia of the ileum with extensive gangrene of the midgut, due probably to a volvulus. The condition in this case was hopeless.

The discouraging results in these last 6 cases led in 1939 to the use in cases 29 and 30 of the procedure suggested by Gage and Ochsner.[4] Both patients died. It is felt that ligation of the cardiac end of the stomach will not accomplish its purpose, as secretions in the lower esophageal segment still can and do drain into the trachea.

In cases 31 and 32 the procedure advocated by Gamble [8] was used, though in case 32 this was modified somewhat after the manner suggested by Leven.[9] That is, the stomach was divided and the gastrostomy tube placed in the distal segment for feeding, and the upper segment of the stomach was exteriorized to allow material in the esophagus to drain to the outside. We were unsuccessful in both cases; again the child in case 32 had in addition to the tracheoesophageal fistula and imperforate anus.

CONCLUSIONS

From the foregoing comments certain conclusions seem justified. Anterior gastrostomy is futile except in cases of anomalies of type 2 and type 3a; that is, it is futile unless the lower segment does not communicate with the trachea. These types are rare; each occurred only once in this series, and it is important to point out that the anomaly in case 14 was thought to be of type 2, but there was in fact a communication with the trachea from the upper esophageal segment, that is, type 3a. Differentiation between type 2 and type 3a is difficult or impossible even if an opaque medium is used at roentgen examination.

Regardless of what procedure is used for the lower segment (other than a direct anastomosis), the upper segment must be treated in some way to prevent aspiration of the overflow secretions from this blind pouch. Constant suction, in my experience, is dangerous, and Leven [9] has also called attention to this. Early exteriorization of this upper segment has been advocated and practiced here without success, though it is possible

11. Churchill, E. D.: Personal communication to the author.

that it would have been successful in cases 9, 11 and 18 had a posterior esophagostomy not been added to the closure of the fistula at the time of the first operation. Exteriorization of the upper segment commits the patient, if he survives, to some form of permanent exterior esophagus.

Treatment of the lower segment except in the very rare type 2 and type 3a anomalies must include measures to prevent contents from the lower segment from entering the trachea. The procedure suggested by Gage and Ochsner,[4] in my experience, does not do this, as tying the cardia leaves undrained (except into the trachea) the secretions of this lower segment. The procedures suggested by Gamble [8] and Leven [9] allow, in theory, free drainage of the secretions of the lower segment of the esophagus. But some method of caring for the secretion from the upper segment is necessary as well as later procedures for constructing an exterior esophagus.

In my experience these methods of Gamble and Leven directed toward exterior drainage of the lower segment have definite disadvantages and dangers. After the procedure is done, if the infant is placed with the head up to allow dependent drainage of the lower segment, the upper segment fills, and the secretions will overflow into the trachea. If the patient is placed with the head lowered, drainage of the upper segment is facilitated, but secretions from the lower segment are allowed to gravitate through the fistula into the trachea. Also, in the 2 cases in which these procedures were used it was noted that there was increased respiratory difficulty, which was thought to be due in part to free escape of the inspired air through the tracheal fistula and out the exteriorized lower segment of the esophagus.

In cases 9, 11, and 18, in which the patients lived fifteen, thirty-two and twenty-eight days respectively after the first operative procedure, it is possible that had the first operation been that of only tying the fistula, instead of in addition freeing the lower segment and suturing it to the back to serve as an esophagostomy for feeding, a recovery might have resulted, though the construction of the undesirable external esophagus would still have been required. In my experience this lower posteriorly exteriorized segment will retract within the chest if not sufficiently freed. If sufficiently freed, the suture line holding the open end to the skin of the back will slough, because the blood supply of the mobilized segment of the esophagus has been interfered with. When such sloughing occurs, mediastinitis is probably unavoidable. Posterior esophagostomy should be abandoned.

No method of attack that does not have as its first step direct closure of the fistula between the lower segment and the trachea has any reasonable chance of success, particularly as regards a primary anastomosis, which is the goal of surgical endeavor in this condition. Direct exposure of the site of the fistula by the extrapleural approach is by no means as

formidable a procedure as has been thought. Indeed, with increasing experience and improvement in technic and anesthesia, it seems to involve less shock—certainly no more—than the methods that include a gastrostomy with anterior drainage of the lower segment of the esophagus.

EXTRAPLEURAL APPROACH TO THE MEDIASTINUM

The patient lies on his face with the head slightly lowered and the right arm extended over the head. The cutaneous incision starts at the level of the first and second ribs, just to the right of the spine and over the transverse processes. This incision runs downward and parallel to the spine to about the level of the fifth rib, where it begins to curve laterally below the scapula and is continued to about the posterior axillary line. The presenting muscles are sectioned and the thoracic cage exposed. The erector spinae muscles are partially sectioned at the level of the fourth rib and pulled medially. The fourth rib is resected subperiosteally for about 1½ to 2 inches (3.7 to 5 cm), starting close to the transverse process. The third and fifth ribs are cut subperiosteally close to the transverse processes. If more room is needed, the sixth rib and occasionally the second may be so treated. A horizontal incision of the intercostal muscle is made just above the bed of the fourth rib, starting from the transverse process and carried laterally for about 2 inches (5 cm.). At the medial end of this incision the intercostal muscles between the third and fourth ribs and between the fourth and fifth ribs are sectioned by vertical incisions. The fourth and usually the third and fifth intercostal nerves have to be sectioned. The horizontal incision is then widened by freeing the muscles from the underlying pleura. With gentleness and care this can be readily done without tearing the pleura.

Starting close to the spinal column, the pleura is pushed laterally and forward so that the mediastinum can be entered. The first landmark is the azygos vein, which will be seen crossing the field horizontally and dorsal to the level of the bifurcation of the trachea. This vein is doubly ligated and cut between the ligatures. Careful exploration of the fatty and areolar tissue adjacent to the bodies of the vertebra will bring into view the lower segment of the esophagus. It is readily identified and freed unless, as in case 25, there happens to be dextraposition of the aorta. The fistula, which is almost invariably at the bifurcation of the trachea, is identified; it is doubly tied with silk close to the trachea, and a cut is made between the ties.

The upper segment of the esophagus is usually to be found just above and dorsal to the bifurcation of the trachea, and it must now be decided whether to proceed with any attempt to free both ends and perform an anastomosis. If the gap is too wide to allow an anastomosis without tension, it is probably futile to attempt it. I have had no experience with

the use of mechanical means, such as a tube of absorbable magnesium, to bridge this gap. Churchill [11] has attempted such a procedure in a few cases but has stated that was not successful and that he has abandoned it.

By using the extrapleural approach it is possible to drain the posterior mediastinum, and drainage is desirable. It is not feasible to drain the mediastinum when using the transpleural approach. With experience it is possible to enter the mediastinum and to perform the anastomosis without opening the pleural cavity. Although resecting one rib and sectioning two others as well as their intercostal nerves takes longer than the intercostal transpleural approach, I do not believe that it adds unduly to the respiratory difficulties after operation or that it is so severe a strain on the patient as the collapse of the right lung which is necessitated by the transpleural approach. The choice between these two approaches is still a question that further experience will help to decide. Churchill [11] has made several attempts via the transpleural approach, but, as in this series, there was a fatal outcome in each.

If infection occurs when the extrapleural approach is used, it is likely that there is a far better chance for it to be taken care of by the extrapleural drainage of the mediastinum. If it occurs when the transpleural approach is used, both the mediastinum and the pleural cavities are likely to be involved, and there is no feasible method of draining either at the time of operation.

Direct exposure of the site of the fistula has many advantages and should be the method of choice. With this approach the exact type of anomaly may be determined and the fistula may be closed. If the distance between the two segments is not too great, a direct anastomosis is possible, as is shown by cases 16, 19, 20 and 22. The patients in cases 20 and 22 lived eight and nine days respectively, and postmortem examination in case 22 showed that death was due to overhydration and not to infection, pneumonia or mediastinitis. That this method will eventually be successful I have no doubt.

As operative procedures in the region of the fistula between the lower segment and the trachea were done in so many cases of this series, it is not possible to give with any accuracy the average distance between the upper and the lower esophageal segment. From the figures as estimated by the operator and the figures compiled from autopsy in nonoperative cases (some of which antedate the period considered in this report) it appears that this distance varies from 0 to over 5 cm., the average distance being approximately 2 cm.

The ideal case in which to attempt a direct anastomosis is one in which the patient is seen early and in which separation of the two segments, as observed at the time of closing the fistula of the lower segment, is 2 cm. or less. In patients coming to operation late and in

poor condition or in whom there is obviously too wide a separation of the segments to justify attempted anastomosis, the procedure should be only that of closing the fistula. Then, just as soon as the infant's condition warrants, and the sooner the better, the upper segment should be exteriorized in the neck. This should be followed by an anterior gastrostomy, and again the sooner the better. Once these procedures are accomplished, months or years may be allowed to elapse before construction of an exterior tube to connect the upper esophagostomy opening with the gastrostomy opening is undertaken.

These procedures, however, are to be avoided when possible, and every reasonable risk should be taken to secure a primary anastomosis. If a direct anastomosis has been done, the problem arises of how best to meet the fluid and caloric needs of the patient until sound healing has occurred. With better understanding of fluid balance, I believe that the patient's nutrition can be maintained by intravenous dextrose and saline solutions supplemented with transfusion of either whole blood or blood serum. Case 22 showed that the danger of overhydration is perhaps as great as that of underhydration. Given another opportunity, I shall attempt to meet the patient's needs without doing a gastrostomy, though such a step can be taken if conditions warrant. A reasonably healthy infant can be maintained under an intelligent regimen of parenteral feeding for a period sufficient to allow healing.

ANESTHESIA

Most of these operations were done with the patient under avertin with amylene hydrate anesthesia (—80 mg. per kilogram) and local infiltration with procaine hydrochloride, supplemented in some cases by oxygen and ether. Recently I have used cyclopropane as a supplement to local procaine hydrochloride anesthesia, and I believe it to be advantageous. For the use of any inhalation anesthesia a well fitting infant's face mask is essential.

SUMMARY

An analysis of 32 cases of congenital atresia of the esophagus is presented. In spite of the fatal outcome in all the 30 operative cases, it is felt that considerable progress along rational lines is being made. The successful operative treatment of a patient with this anomaly is only a question of time.

For a successful outcome, prompt recognition of the condition during the first few hours of life is essential. It is perhaps not advisable to go so far as to insist on routine examination of the esophagus of every infant by the passing of a catheter, as has been suggested by Gage and Ochsner,[4] but it is imperative that any infant who exhibits any of the characteristic signs or symptoms of esophageal atresia should at once have a catheter

passed down the esophagus, and roentgenograms should be taken without the use of any opaque medium. Once the obstruction is discovered, immediate operation should be undertaken.

An associated tracheoesophageal fistula was present in 91 per cent of this series. A direct attack on the tracheoesophageael fistula, if one is present, should be undertaken. From the experience presented here, the extrapleural approach would seem to be safer than the transpleural.

If direct anastomosis is possible, it should be done. If it is successful and no complication develops, the problem for that case is solved, barring possible esophageal stenosis later. If direct anastomosis of the two segments appears impossible or inadvisable, the tracheoesophageal fistula should be closed and nothing further done at that time. Subsequent procedures to be done just as soon as conditions permit are, first, the exteriorization of the upper esophageal segment and, second, an anterior gastrostomy. If the patient survives, construction of an exterior connection between the upper esophagostomy and the anterior gastrostomy can then be postponed until a suitable age. One must remember that an exterior esophagus as a palliative procedure for an elderly patient with cancer is justifiable and endurable, but one dreads to commit an infant to the sort of existence it entails. Every effort should be made to recognize these conditions in the first few hours of life as well as to improve surgical technic to such a degree that a direct anastomosis will be possible in an increasing number of cases.

Given a suitable case in which the patient is seen early, I feel that, with greater experience, improved technic and good luck, the successful outcome of a direct anastomosis can and will be reported in the near future.

ROENTGEN DEMONSTRATION OF ESOPHAGEAL VARICES

ITS CLINICAL IMPORTANCE

RICHARD SCHATZKI, M.D.

BOSTON

Twelve years have passed since Wolf [1] reported 2 cases in which he had demonstrated varices of the esophagus roentgenologically. His original observations have been confirmed and amplified by others.[2] In 1931 I published 5 cases and described for the first time the roentgen demonstration of varices of the stomach.[3] A report on 45 cases appeared three years later.[4] Numerous cases have since been observed by many roentgenologists. Nevertheless, a systematic and routine search for varices, particularly for those of minor degree, has not been widely practiced. In addition, the clinical importance of roentgen demonstration of varices has not received the recognition which it deserves. This is apparent when one reads publications from large teaching centers in which the differential diagnosis of splenomegaly is extensively discussed without mention of roentgen visualization of varices. It seems justifiable, therefore, to review the roentgen demonstration of varices and to discuss its clinical value.

This review is based on a series of 116 cases in which I have demonstrated esophageal varices.

The principle of roentgen visualization of esophageal varices is simple. It rests on the fact that the dilated veins bulge into the lumen and produce an uneven, wormlike surface of the inside of the esophagus. These

---

From the Department of Roentgenology, the Massachusetts General Hospital.

1. Wolf, G.: Erkennung von Oesophagusvarizen im Röntgenbild, Fortschr. a. d. Geb. d. Röntgenstrahlen **37**:890-893, 1928.

2. Berg, H. H.: Röntgenuntersuchungen am Innenrelief des Verdauungskanals, ed. 2, Leipzig, Georg Thieme, 1931. Hjelm, R.: Zwei Fälle von röntgendiagnostizierten Oesophagusvarizen, Acta radiol. **12**:146-150, 1931. Kirklin, B. R., and Moersch, H. I.: Report of a Case of Roentgenologically Demonstrable Esophageal Varices Complicating Splenomegaly, Radiology **17**:573-575, 1931. Beutel, A.: Oesophagusvarizen, Acta radiol. **12**:527-532, 1932. Oppenheimer, A.: Esophageal Varices, Am. J. Roentgenol. **38**:403-414, 1937.

3. Schatzki, R.: Die Röntgendiagnose der Oesophagus- und Magenvarizen und ihre Bedeutung für die Klinik, Fortschr. a. d. Geb. d. Röntgenstrahlen **44**: 28-39, 1931.

4. Schatzki, R.: Reliefstudien an der normalen und krankhaft veränderten Speiseröhre, Acta radiol., 1933, supp. 18, pp. 1-149.

Fig. 1.—Extensive esophageal varices in a patient with cirrhosis of the liver. *A*, right anterior oblique view, showing several vessels superimposed on each other. *B*, left anterior oblique view. A large isolated tortuous vessel is demonstrated.

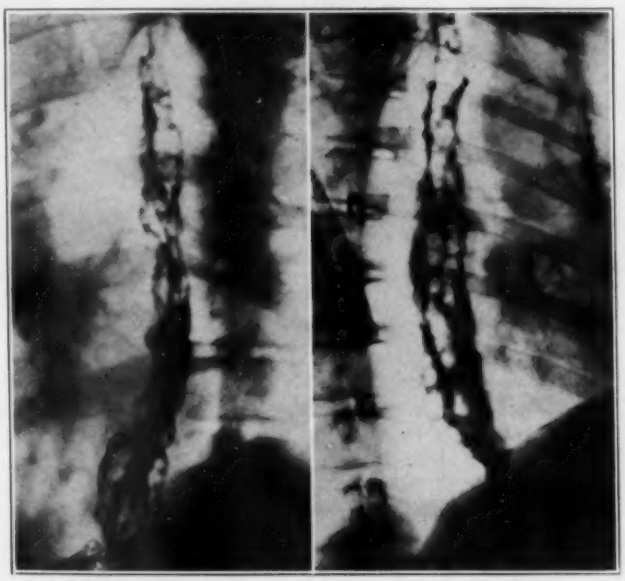


Fig. 2.—Extensive varices in a patient with cirrhosis of the liver. The patient was clinically thought to have an intestinal tumor. See data in text (case 3).

protruding structures will be completely obliterated if one fills the organ with a large amount of barium sulfate. If, however, one coats the inner surface of the esophagus with only a thin layer of barium, the dilated vessels will be visible. The success of the examination depends on the technic used to obtain the desired degree of coating. This is especially true in those cases in which the varices are comparatively small.

TECHNIC

A watery suspension of barium sulfate serves as contrast substance. It contains equal amounts by volume of barium sulfate and water and

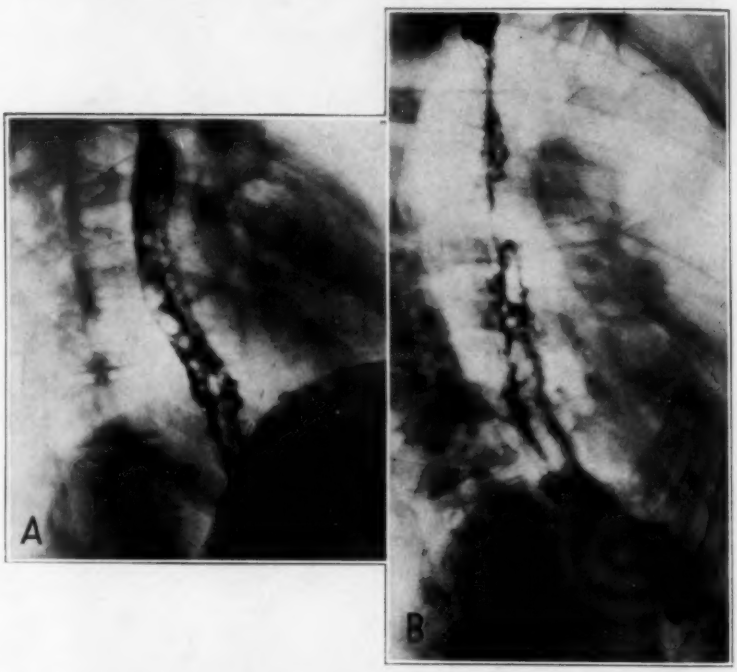


Fig. 3.—*A*, extensive varices in a woman with Banti's syndrome. The patient was 20 years old, with an eight year story of repeated fever, anemia and pain in the left upper abdominal quadrant. There was splenomegaly. The clinical differential diagnosis was between lymphoma and Banti's syndrome. It was decided in favor of Banti's syndrome by the demonstration of varices. Splenectomy was performed. Eight months later there was a first, gross and fatal hemorrhage. *B*, extensive varices in a patient with cirrhosis of the liver. Examination was done in order to determine the clinically uncertain source of hematemesis. There was no ascites, and the spleen was not palpable.

is the same suspension used in the routine examination of the gastrointestinal tract. Occasionally a slightly thicker mixture is required to obtain the necessary coating. The use of atropine, as previously advised,[4]

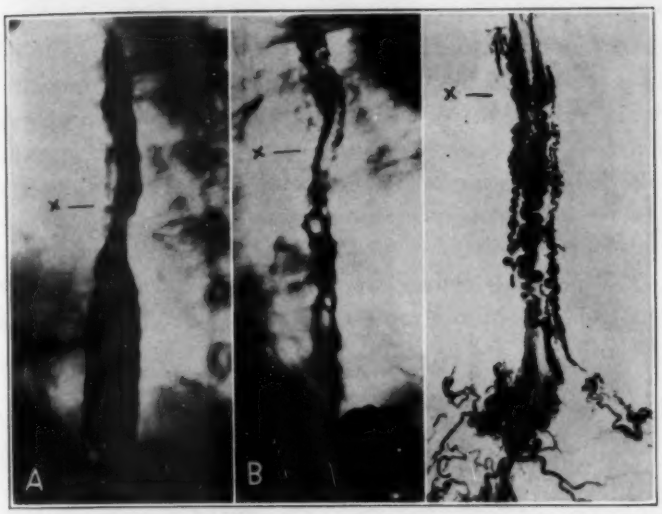


Fig. 4.—Extensive varices in a patient with cirrhosis of the liver. *A*, complete filling of the esophagus shows widening of the organ, with localized narrowing at *x*. *B*, the relief picture shows extensive varices which are superimposed on each other in the lower half of the esophagus. Two large vessels are visible at *x*. *C*, roentgenogram of the injected opened specimen.

Fig. 5.—Marked dilatation of the esophagus due to varices in a patient with cirrhosis. The varices extend through the cardia. There was marked delay in the emptying of the esophagus, but no dysphagia.

is rarely necessary. The moment of optimum coating is different in every case. Often it occurs at the end of deglutition, but in other instances it is obtained by waiting for a small amount of barium to flow back into the esophagus from the stomach. The latter technic is especially useful in cases of questionable small varices. The demonstrability of varices depends on a number of other factors, which will be discussed later (gravity, peristalsis, projection and respiration).

Fluoroscopic examination is of great importance in the discovery of varices. Only by this method can the optimal degree of coating of the esophagus and the optimal projections, which vary from case to case,

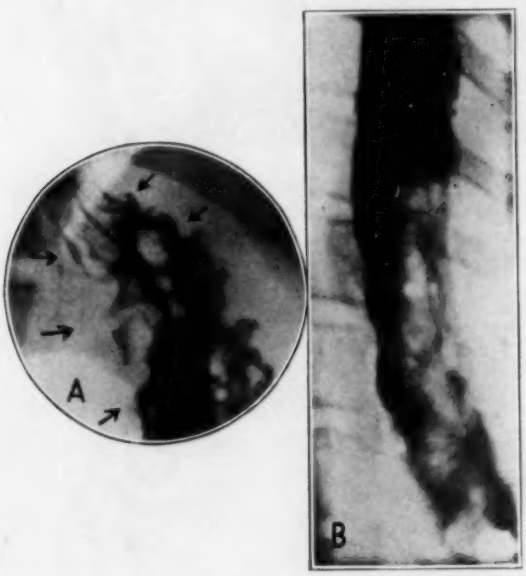


Fig. 6.—*A*, varices of the stomach close to the cardia (see arrows) in a patient with esophageal varices. *B*, large, tortuous, isolated vessel in the lower portion of the esophagus in a boy of 13 with Banti's syndrome. There had been a recent hemorrhage. The patient died shortly afterward from mesenteric thrombosis. Autopsy showed thrombosis of the splenic vein and of the superior mesenteric vein.

be decided. Extensive varices can easily be diagnosed during fluoroscopic study, but in the demonstration of small or questionable ones roentgenograms are indispensable. In such cases the fluoroscopic examination may give completely negative results, while the roentgenograms show the presence of varices. Roentgenograms are best taken during fluoroscopic examination (the so-called "spot," or aimed, films). Whenever the available equipment does not permit such a procedure, films

taken on the Bucky table in a projection and at an interval after deglutition determined by a preceding fluoroscopic examination will be a useful alternative.

ROENTGEN APPEARANCE OF VARICES

Varices may be visible in a completely filled esophagus if they run along the projected edge, wherein they produce a notched or scalloped appearance. This profile picture, however, is usually not reliable. It is necessary to obtain a relief view of the varices. In the presence of varices the normal mucosal relief picture of the esophagus with its parallel longitudinal folds is changed. There results an intricate and

Fig. 7.—*A*, isolated enlarged vein, which is only slightly tortuous, in a case of biliary cirrhosis. *B*, small varicose vessels in a case of cirrhosis of the liver. The patient had his first hemorrhage two years after this examination.

often network-like pattern which is due to superimposition of the vessels bulging from the opposing walls of the esophagus (fig. 4). By rotating the patient it is usually possible to demonstrate one or more isolated tortuous vessels (figs. 1, 6*B*, 7*A*, 8 and 12). In a given case the dilated vessels have a definite configuration and arrangement, although their size may change considerably (see section on the influence of various factors on the demonstrability of varices).

Varices, if present, are with extremely rare exceptions located in the lower portion of the esophagus. They are often not confined to this area but may extend upward to involve the entire thoracic portion and

occasionally the cervical portion of the esophagus. However, if this is the case the esophagus immediately above the diaphragm is always involved, and the examiner should therefore concentrate on this area.

I have observed a few cases in which the varices extended into the stomach, where they produced an enlargement of the tortuous gastric folds similar to that seen in cases of localized gastritis (fig. 6 *A*). These changes were always confined to an area close to the cardia, with 1 exception, in which the varices were localized in the extreme end of the gastric fundus. No esophageal varices were present in this particular case. My observations have since been confirmed by Pape.[5]

Fig. 8.—Varices localized in the lower portion of the esophagus in a young woman with cirrhosis of the liver. The clinical differential diagnosis was between tuberculous peritonitis and cirrhosis. The patient died from insufficiency of the liver shortly afterward. *A*, right oblique view. The arrow indicates the area in which the normal folds end and the varices begin. *B*, left oblique view.

The large diameter of the esophagus is striking in many cases of varices. It is produced largely by protruding vascular structures, which by filling the lumen distend the esophageal walls. A picture similar to that of idiopathic dilatation of the esophagus may result (fig. 5). There is, however, no obstruction at the cardia. The barium passes freely into the stomach, although the emptying time and particularly the "cleaning"

5. Pape, R.: Ueber Deformationen des Magenfornix bei Oesophagusvarizen, Röntgenpraxis **9**:809-813, 1937.

time of the esophagus may be markedly prolonged. The discovery of a wide esophagus in a young person, particularly if the dilatation is localized to the lower end, demands a search for varices.

INFLUENCE OF VARIOUS FACTORS ON DEMONSTRABILITY OF VARICES

1. *Gravity.*—Varices are usually much smaller with the patient in the upright than in the horizontal position. Therefore, the examination should be performed with the patient in the horizontal position.

2. *Peristalsis.*—Peristalsis (figs. 10 and 11) not infrequently decreases the size of the dilated vessels. At times one sees even large

Fig. 9.—Varices in a localized area in the lower end of the esophagus. The patient had albuminuria, hematuria and ascites. There was questionable enlargement of the spleen, not confirmed by all examiners. A tentative clinical diagnosis was chronic nephritis and "old malaria spleen." The demonstration of varices proved the presence of obstruction in the portal system. Splenectomy showed the presence of thrombophlebitis of the splenic vein.

varices empty completely during contraction of the esophagus, and a normal relief may result locally. Therefore, the presence of normal mucosal folds during the phase of contraction does not prove the absence of varices. The influence of peristalsis on the size of the varices apparently varies from case to case but seems to be fairly characteristic for a given case.

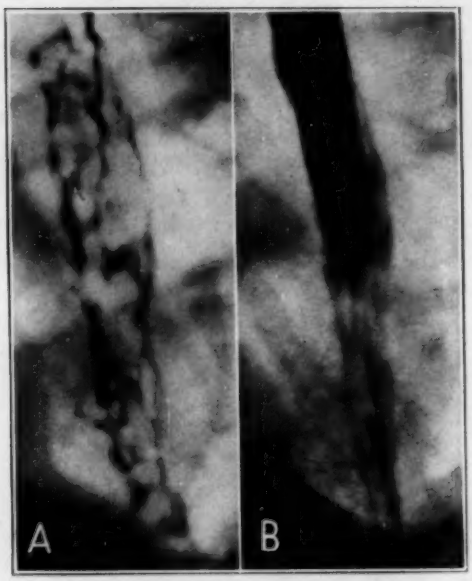


Fig. 10.—Changing size of varices during peristalsis in a patient with Banti's syndrome. The two pictures were taken within a few minutes of each other, with identical position of the patient. *A*, extensive varices during the resting phase of the esophagus. *B*, emptying of most varices during the contraction of the esophagus.

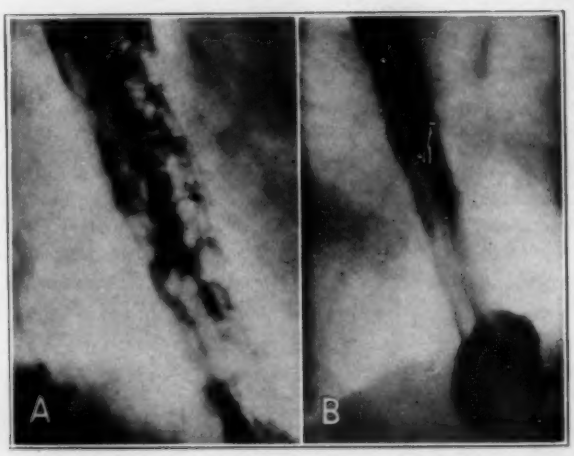


Fig. 11.—Decrease in size of the varicose veins during peristalsis in a patient with cirrhosis of the liver. *A*, phase of rest of the esophagus. *B*, phase of contraction.

3. *Projection.*—If only one enlarged vessel is present, it may, like polarized light, undulate in one plane exclusively (fig. 12). In such a case the characteristic sign of the vessel, namely its tortuosity, is absent if the examination is performed in the direction of this plane. On the other hand, the vessel becomes obvious if the roentgen rays are perpendicular to the plane in which the vessel swings. The plane of undulation varies from case to case and cannot be predicted. For this reason it is necessary to examine the esophagus routinely in several projections and to take films in at least two projections which are about

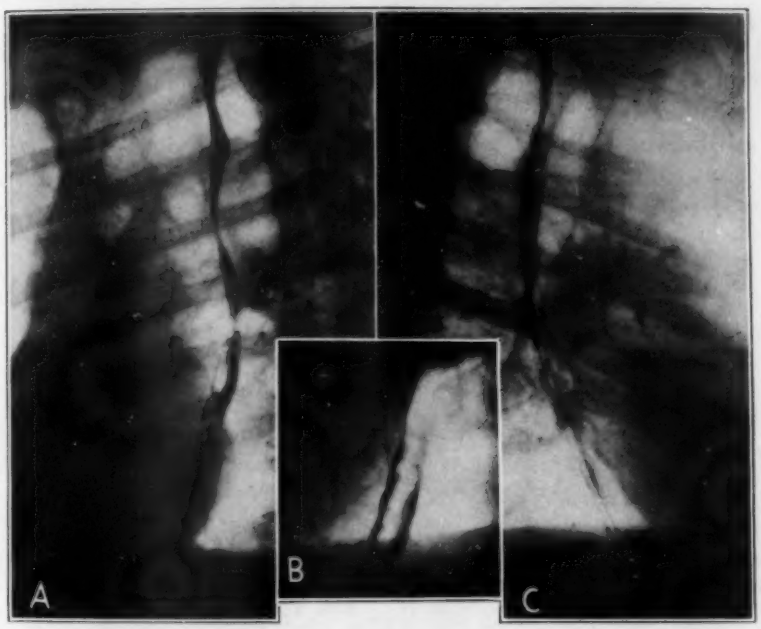


Fig. 12.—Influence of the projection on the demonstrability of a varicose vein. The tortuous vein undulates in one plane only. The vessel in this case is not visible in the right anterior oblique view (*C*), while it is clearly seen in the left anterior oblique view (*A* and *B*) and particularly well on the spot film in this projection (*B*).

90 degrees different from each other. As the phenomenon described is particularly common in the presence of small varices, such views are of special importance in those cases in which fluoroscopic examination reveals no abnormality.

4. *Respiration.*—Quiet respiration does not noticeably influence the enlarged vessels, but deep inspiration and expiration at times change their size. They may be slightly smaller with inspiration and larger with expiration. These findings are by no means constant, since the

influence of the changing intrathoracic pressure during respiration is rather complicated. I have previously described [4] a test in which changes in intrathoracic pressure were used for demonstration of changes in the size of the varices. Marked differences in pressure were produced by employing the Valsalva test (expiratory effort with closed glottis after deep inspiration) and the Mueller test (inspiratory effort with closed glottis after deep expiration). At times an increase in the size of the vessels was observed following the Valsalva test, while at other times such an increase followed the Mueller test. The variable results are apparently due to the fact that the size of the vessels depends on two factors: (1) the increased pressure in the thorax and therewith in the esophagus, which tends to compress the vessels during the Valsalva test, and (2) the increased pressure which exists during the Valsalva test within the vascular system proximal to the anemic lungs, as a result of which the varices tend to be enlarged. This test is of some theoretic interest, but in practice it has not been of great help, partly owing to the fact that only a few patients show sufficient intelligence and cooperation for its performance. In the routine examination I believe it best to take films after slight inspiration, since during this phase the lower end of the esophagus is stretched slightly, thus obviating the possibility of misinterpreting tortuous folds in a slack esophagus.

DIFFERENTIAL DIAGNOSIS

In cases of extensive varicosities the roentgen appearance of the esophagus is usually so characteristic that differential diagnostic difficulties rarely arise. The situation is different when the varices are small and localized. Air bubbles in the esophagus may occasionally assume the shape of small varices, in which case reexamination prevents error.

Slightly tortuous folds, which occur not uncommonly in the lower part of the esophagus, especially in elderly persons, simulate small varices. The latter are usually slightly larger in caliber than the tortuous folds, and in addition the size of one dilated vessel usually contrasts with the narrower normal folds. Films taken during inspiration, which stretches the tortuous folds of the esophagus, may be of help in differential diagnosis. The misinterpretation of tortuous gastric folds in a small hiatus hernia of the stomach may be prevented by adequate filling of the esophagus and stomach with barium.

A more important problem is the differential diagnosis between varices and "curling" of the esophagus (figs. 13 and 14). "Curling" [4] is a peculiar phenomenon seen occasionally during deglutition, in which, during the contraction, multiple irregular toothlike projections are seen along the edges of the esophagus. If closely spaced they may produce a scalloped appearance of the organ. The phenomenon appears and disap-

pears rapidly, but it can usually be easily reproduced by letting the patient swallow again, with or without additional barium. I believe, with Fleischner,[6] that the picture is produced by multiple adhesions of the esophageal wall which prevent sufficient collapse of the organ during contraction. Clinical symptoms are apparently not present; nevertheless, the recognition of "curling" is important for reasons of differential diagnosis. Since the profile picture of varices may be similar to that of "curling," it is necessary to rely for diagnosis on the relief picture, which is distinctly different.

The most important diagnostic question from the clinical point of view is the differentiation between varices and cancer of the esophagus.

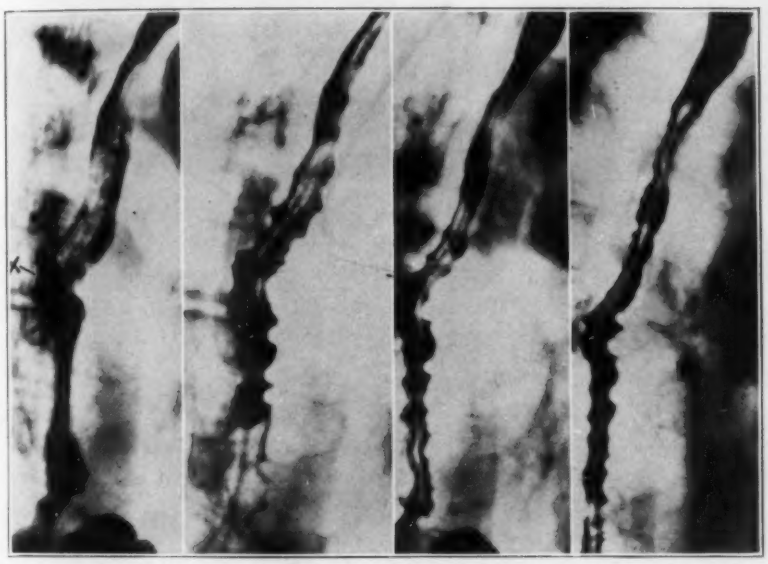


Fig. 13.—"Curling" of the esophagus, probably produced by multiple areas of adhesions. Note the definite point of adhesion at *x*.

The marked irregularity of the inner relief in cases of extensive varices produces a picture which occasionally closely simulates that of cancer (Pohlandt[7]).

Since for all practical purposes, if varices are present they are always immediately above the diaphragm, they must be differentiated from cancer in this region only. The exact roentgen study of the lesion in most cases readily outlines the cancer, with its sharp edges and its ulcer-

6. Fleischner, F.: Die Divertikel der Speiseröhre. Haft-oder Adhäsionsdivertikel, Fortschr. a. d. Geb. d. Röntgenstrahlen **45**:627-664, 1932.

7. Pohlandt, K.: Oesophagusvarizen oder Carcinom? Röntgenpraxis **3**:889-895, 1931.

ation, in contrast to the nonulcerated, poorly defined defect produced by varices. Of great help is the study of the elasticity of the esophagus. The esophagus becomes rigid in most cases of cancer, but it never loses

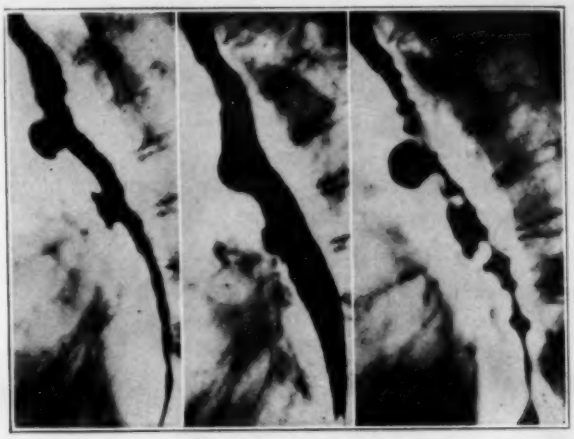


Fig. 14.—Adhesion diverticula, together with "curling."

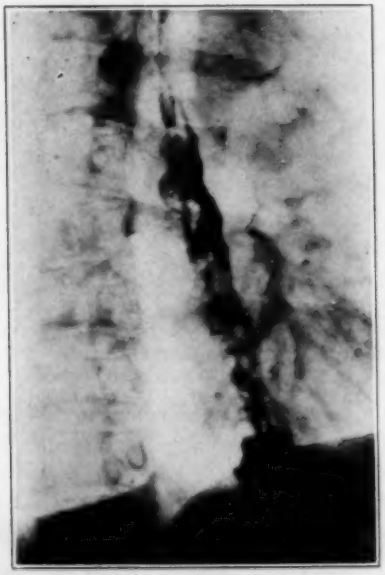


Fig. 15.—Extensive carcinoma of the esophagus, simulating varices. The patient had dysphagia, which was distinctly against the diagnosis of varices.

its elasticity with the most extensive varices. The organ can be easily widened by a large amount of barium, and it appears flexible during peristalsis and respiration and with the pulsation of the heart. The

varices produce no obstruction, though emptying of the esophagus may be markedly delayed by the pronounced irregularities of the inner surface. The disappearance of varices during peristalsis may occasionally be the deciding differential point. Usually, however, the localization, the appearance and the elasticity of the questionable area allow a ready differentiation between the two lesions. Should all these signs become unreliable in a rare case, a clinical symptom, namely, dysphagia, may be of decisive importance. Contrary to some reports in the literature (Oppenheimer [2]), I have never seen dysphagia in cases of the most extensive varices, even when the emptying time of the esophagus was markedly prolonged (more than two hours in 1 of these cases). Hence, difficulties in swallowing should not be explained on the basis of varices (fig. 15).

CLINICAL IMPORTANCE OF ROENTGEN DEMONSTRATION OF VARICES

Congenital varices have been described in rare cases. A slight degree of telangiectasia, usually in the upper portion of the esophagus, has been found in elderly persons in whom there was no evidence of portal obstruction (Kaufmann [8]). Such cases are exceptions. For all practical purposes, the roentgen demonstration of varices in the lower end of the esophagus proves the presence of obstruction in the portal circulation. In all my cases in which postmortem examination was done, either cirrhosis of the liver or thrombosis of the splenic vein was found to account for the varicose collateral vessels. I have not observed a proved case in which pressure on the portal vein or its branches by tumor masses produced esophageal varices.

The clinical picture of cirrhosis of the liver may be classic, and then the demonstration of varices furnishes only confirmatory evidence. In other cases the diagnosis is suspected but becomes certain with the visualization of varices. Finally, there are those cases in which the diagnosis is not even entertained. In this connection, I have seen patients with esophageal varices in whom the liver and spleen were not palpable and in whom demonstrable ascites was absent; in other words, patients in whom the varices were the only demonstrable anatomic evidence of cirrhosis of the liver. The demonstration of varices has always proved helpful regardless of whether the diagnosis of cirrhosis was unsuspected, suspected or believed to be certain prior to roentgen examination. The first case to be reported may serve as an example.

---

8. Kaufmann, E.: Pathology for Students and Practitioners, translated by S. P. Reimann, Philadelphia, P. Blakiston's Son & Co., 1929.

REPORT OF CASES

CASE 1.—An Italian woman 44 years old entered the hospital because of swelling of the ankles and slight dyspnea on exertion. Physical examination showed a large abdomen with questionable free fluid. There was a tender epigastric mass in addition to a questionable mass in the left upper quadrant. The latter was not felt by all the examiners. Pelvic examination showed masses in both lateral vaults, which were tender to palpation. The clinical impression was that of malignant tumor arising in the pelvic organs, with metastases to the liver, lung and brain. Lymphoma was thought another possibility. Roentgen examination showed varices in the lower third of the esophagus and the probable presence of a small liver and an enlarged spleen. Hepatic damage was then confirmed by positive results from Takata-Ara and bromsulfalein tests (90 per cent retention in one hour). At discharge, the masses in the upper part of the abdomen were thought to be the liver and spleen. The patient returned to the hospital one year later, with a similar picture.

Not all patients with cirrhosis show varices roentgenologically. In a previous statistical analysis [4] I found that varices were demonstrable in about 50 per cent of all cases of cirrhosis and that the percentage was higher in cases of advanced involvement with associated splenomegaly, ascites or hematemesis.

Varices are even more frequently seen in patients who present the so-called Banti's syndrome. At the Massachusetts General Hospital, 15 patients were examined roentgenologically in the last six years in whose cases the final clinical diagnosis was Banti's syndrome. In 12 of these patients the presence of congestion within the portal system was confirmed by operation or autopsy. Of the 3 who came to autopsy, 2 had thrombosis of the splenic vein, and 1 had cirrhosis of the liver. In 3 of the 15 cases the diagnosis was based on clinical evidence only. Varices were observed roentgenologically in 13 of these patients. One of the 2 in whose cases roentgen study gave negative results was a young child. The clinical diagnosis was considered doubtful at discharge, and an operation was not performed. The other was a patient with repeated hematemesis. At operation markedly dilated veins were found on the outside of the stomach, together with slight enlargement of the spleen. It will be seen that, in my material, varices were demonstrable in nearly all cases of Banti's syndrome. The varices in this group are usually very extensive [9] (figs. 3*A*, 6*B* and 10), small localized varicosities being less commonly seen (fig. 9).

In my experience the demonstration of varices has proved to be of importance also in the diagnosis of primary carcinoma of the liver,

9. Brdiczka, I. G., and Tschakert, J.: Die röntgenologische Diagnostik der Oesophagusvarizen, Fortschr. a. d. Geb. d. Röntgenstrahlen **46**:156-166, 1932. Plotz, M., and Reich, N. E.: Esophageal Varices in Portal Hypertension (Pathogenesis and Diagnosis by Roentgenography), Am. J. Digest. Dis. **5**:357-360, 1938. Greenwald, H. M., and Wasch, N. J.: Roentgenological Demonstration of Varices as Diagnostic Aid in Chronic Thrombosis of Splenic Vein, J. Pediat. **14**:57-65, 1939.

owing to the fact that this disease is usually associated with cirrhosis of the liver (in 87 per cent of the cases from the Massachusetts General Hospital [10]). The demonstration of esophageal varices in a patient who is known to have malignant disease (e. g., pulmonary metastasis) and who has a mass in the region of the liver is evidence highly suggestive of the presence of primary carcinoma of the liver.

Visualization of varices offers much help in cases of hematemesis. Establishment of the source of bleeding is important for diagnosis, prognosis and treatment. Roentgen recognition of varices has decreased the number of cases of so-called roentgen-negative hematemesis. Thereby, unnecessary exploratory laparotomies performed in a search for the bleeding point and prolonged medical dietary regimens for hypothetic bleeding ulcers have been avoided. Furthermore, the prognostic importance of the demonstration of varicosities was impressive in a number of my cases in which the patients died of hemorrhage weeks or months after the examination. In some of these cases the hemorrhage was the first and only one.

The discovery of varices is valuable in differentiating the causes of ascites. I have seen patients with ascites in whose cases the suspected clinical diagnosis was abdominal malignant tumor and others in whose cases the possibility of cardiac ascites was discussed, while 1 patient was thought to have tuberculous peritonitis. In all these cases the demonstration of varices proved that the ascites was due to portal obstruction, i.e., cirrhosis of the liver.

The diagnostic importance of the demonstration of varices in patients with splenomegaly cannot be overemphasized. I have had several cases in which visualization of esophageal varices was the decisive diagnostic step whereby lymphoblastoma, etc., as a cause of the splenomegaly was excluded. The following case will serve as an example.

Case 2.—A man 58 years old complained of anorexia and loss of weight for six months. The liver was found to be enlarged. There was a mass in the left upper quadrant of the abdomen, the nature of which was debated. The house staff favored a diagnosis of malignant tumor, and one of the visiting physicians diagnosed cirrhosis of the liver, while two others made the presumptive diagnosis of lymphoma. Lymphoma was considered to be a definite possibility even after roentgen demonstration of varices had been carried out. Roentgen ray treatment as a therapeutic test was advised and started. As there was still much clinical debate about the nature of the mass, a laparotomy was finally decided on. It confirmed the roentgen diagnosis of cirrhosis. This case goes back a number of years to a time at which roentgen diagnosis of varices and its consequences were not fully appreciated. At present the radiation test and an exploratory laparotomy in such a case would be considered unnecessary and even contraindicated.

10. Schatzki, R.: Roentgenological Diagnosis of Primary Carcinoma of the Liver, to be published.

Finally, I have in a few cases found the demonstration of varices helpful in the differential diagnosis of a mass in the left upper quadrant of the abdomen. In such a case, if varices are present, the mass in all probability represents an enlarged spleen.

CASE 3.—A man 52 years old, with a history of rectal bleeding for four years, showed a palpable mass in the upper part of the abdomen. He was given a barium sulfate enema, and a roentgen study of the upper part of the gastrointestinal tract was made to establish the location of the supposed intestinal tumor. Roentgen examination showed the mass to be outside the gastrointestinal tract and to represent an enlarged, lobulated spleen. The presence of extensive esophageal varices established the existence of portal obstruction (fig. 2).

Roentgen demonstration of varices is no longer a rare curiosity but is an important part of the daily routine examination of the gastrointestinal tract.

SUMMARY

The roentgen diagnosis of esophageal varices is discussed, based on examination in 116 cases of varices.

The technic of demonstrating varices and their roentgen appearance are described.

Stress is laid on the visualization of small localized varices, for which a combination of fluoroscopic and roentgenographic methods is essential.

Factors influencing the demonstrability of varices are the degree of filling of the esophagus, gravity, peristalsis, projection and respiration.

Unless these factors are considered, a normal appearance of the esophagus may result even in cases of extensive varicosities.

Differentiation between varices and air bubbles, tortuous normal folds, small hiatus hernia, "curling" and cancer is discussed.

The demonstration of varices is of clinical importance in the diagnosis of cirrhosis of the liver, of Banti's syndrome and of primary carcinoma of the liver. It is of great help in the differential diagnosis of hematemesis, acites and splenomegaly.

BLEEDING ESOPHAGEAL VARICES

AN EVALUATION OF METHODS DIRECTED TOWARD THEIR CONTROL, ESPECIALLY BY DIRECT INJECTION OF A SCLEROSING SOLUTION

WALTMAN WALTERS, M.D.

HERMAN J. MOERSCH, M.D.

AND

D. ANGUS McKINNON, M.D.

Fellow in Surgery, the Mayo Foundation

ROCHESTER, MINN.

Esophageal varices develop as a result of obstruction of the portal or splenic veins because the esophageal veins are one of the three sites of communication between the portal and caval systems. Since the veins in the submucosa of the lower part of the esophagus are poorly supported by loose connective tissue, with the increase in the quantity of blood passing through them varices develop, become superficial to esophageal mucosa and are prone to rupture.

The frequency with which bleeding occurs from esophageal varices was emphasized by Preble's [1] review of 60 cases of fatal gastrointestinal bleeding in 80 per cent of which esophageal varices were found. In 50 per cent of the cases in which varices were present, macroscopic demonstration of the site of perforation of the varix was possible. Rivers and Wilbur [2] found that in 5 per cent of a group of 668 patients with a history of hematemesis bleeding was attributable to cirrhosis of the liver or to splenic anemia.

Methods directed toward prevention of fatal bleeding have consisted of (1) splenectomy to reduce the blood entering the portal vein, (2) omentopexy to establish collateral circulation around the liver through the veins of the peritoneum and the abdominal wall, especially through the superior epigastric veins, (3) interruption of the blood flow through the esophageal veins by ligation of the coronary vein, with or without

---

From the Division of Surgery (Dr. Walters) and the Division of Medicine (Dr. Moersch), the Mayo Clinic.

1. Preble, R. B.: Conclusions Based on Sixty Cases of Fatal Gastro-Intestinal Hemorrhage Due to Cirrhosis of the Liver, Am. J. M. Sc. **119**:263-280 (March) 1900.

2. Rivers, A. B., and Wilbur, D. L.: The Diagnostic Significance of Hematemesis, J. A. M. A. **98**:1629-1631 (May 7) 1932.

splenectomy and, more recently, (4) injection of a sclerosing solution transperitoneally into the paraesophageal veins and directly into the varices through the esophagus.

ETIOLOGY

The factor underlying the development of esophageal varices is not always clear. While Banti's disease and splenic anemia are the conditions most frequently associated with the development of esophageal varices, they cannot, as Dock and Warthin [3] have pointed out, be regarded as distinct clinical entities but may occur as a result of obstruction of the portal or the splenic vein. Many hypotheses have been advanced to account for obstruction of the portal or the splenic vein, but they will not be discussed in this presentation. The physiologic changes involved in the development of esophageal varices have been well demonstrated in the anatomic studies of McIndoe [4] and of Kegaries [5] (fig. 1). Obstruction of the veins may be produced by either intrinsic or extrinsic factors. With the development of obstruction of the portal or of the splenic vein, there is an attempt on the part of nature to establish an anastomosis by means of which the blood from the portal vein can return to the general systemic circulation. This, as a rule, will take place through channels of communication that already exist. The route to be selected is somewhat dependent on the site of the obstruction in the splenic or portal vein. According to McIndoe,[4] the communication that will be established usually takes place through one of three sites (fig. 2):

1. At the point of transition between absorbing and protective epithelium. (*a*) Between the coronary vein of the stomach and the intercostal, azygos and diaphragmatic veins. (*b*) Between the superior hemorrhoidal and the middle and inferior hemorrhoidal veins.

2. At the site of embryologic circulation—the falciform ligament containing the paraumbilical veins.

3. At all situations within the abdomen where the gastrointestinal tract, its appendages or glands developed from it become retroperitoneal developmentally or adherent to the abdominal wall pathologically.

The communications that occur between the coronary vein of the stomach and the azygos, intercostal and diaphragmatic veins are of

3. Dock, G., and Warthin, A. S.: A Clinical and Pathological Study of Two Cases of Splenic Anaemia, with Early and Late Stages of Cirrhosis, Am. J. M. Sc. **127**:24-55 (Jan.) 1904.

4. McIndoe, A. H.: Vascular Lesions of Portal Cirrhosis, Arch. Path. **5**:23-42 (Jan.) 1928.

5. Kegaries, D. L.: (*a*) The Venous Plexus of the Oesophagus, Surg., Gynec. & Obst. **58**:46-51 (Jan.) 1934; (*b*) The Venous Plexus of the Esophagus: Its Pathologic and Clinical Significance, Proc. Staff Meet., Mayo Clin. **8**:160-163 (March 15) 1933. Walters, W., in discussion on Kegaries.

the greatest importance, for they are primarily concerned in the development of esophageal varices. McIndoe has pointed out that in the presence of obstruction of the portal vein these communications are most likely to develop, as they are the most direct and the shortest route between the portal and the general systemic circulation. Furthermore, in the presence of obstruction of the portal vein the pressure within the portal circulation is increased, and in the absence of valves the flow of blood through the coronary vein is undoubtedly reversed and thus readily exerts pressure on the esophageal plexus. Kegaries [5b] has clearly demon-

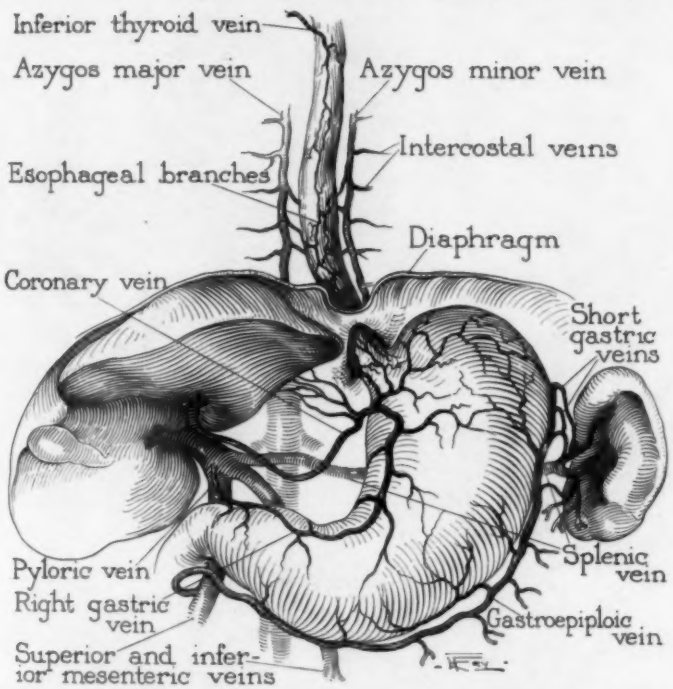


Fig. 1.—Relation of the coronary vein of the stomach to the esophageal veins. (From Kegaries, D. L.: The Venous Plexus of the Oesophagus, Surg., Gynec. & Obst. **58**:46-51 [Jan.] 1934.)

strated the manner in which the anastomosis takes place between the portal and the caval circulation through the splenic veins in the gastrolienal ligament by way of the cardia and the lower part of the esophagus. The coronary vein of the stomach branches as it pierces the muscular coats to reach the submucosa of the cardia. In this region the branches of the coronary vein break up into a number of small longitudinal venules which have no cross communication for a distance of approximately 3 or 4 cm. At this point they break up into a rich plexus, which anastomoses freely with branches of the splenic vein entering the stomach

through the short gastric veins. The larger trunks are extremely tortuous and run in a longitudinal direction. This plexus of veins communicates by perforating branches with larger periesophageal trunks, which likewise run in a longitudinal direction and usually number three or four. The latter trunks communicate with the azygos, the intercostal and the diaphragmatic veins of the systemic circulation (fig. 3).

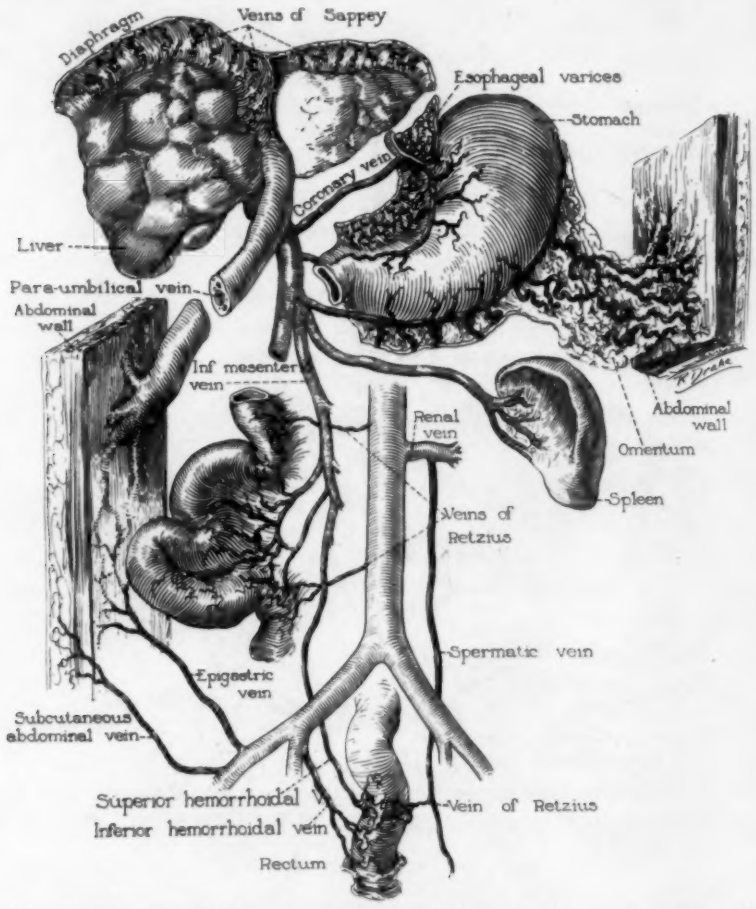


Fig. 2.—The most likely source of communication between the portal and the systemic circulation. (From McIndoe, A. H.: Vascular Lesions of Portal Cirrhosis, Arch. Path. **5**:23-42 [Jan.] 1928.)

The veins in the submucosa of the lower part of the esophagus are poorly supported by loose connective tissue. Because of their situation they are constantly exposed to trauma and consequently most prone to lead to fatal bleeding. In addition, there is the effect of the changing suction which takes place in the thoracic cage with respiration. Eso-

phageal varices may involve the greater part of the esophagus but usually are limited to the lower half.

DIAGNOSIS

One of the greatest obstacles to the development of procedures for the treatment of esophageal varices has been inability to make an early

Fig. 3.—Filling of esophageal venules following injection through the coronary vein. (From Kegaries, D. L.: The Venous Plexus of the Oesophagus, Surg., Gynec. & Obst. **58**:46-51 [Jan.] 1934.)

diagnosis. In 1925, Jackson and his associates [6] called attention to the possible value of esophagoscopic study as an aid in the diagnosis of

6. Jackson, C.; Tucker, G.; Clerf, L. H.; Lukens, R. M., and Moore, W. F.: Hematemesis: A Plea for Objective Methods of Diagnosis, J. A. M. A. **85**: 870-875 (Sept. 19) 1925.

esophageal varices. The procedure did not come into general use, however, because of the fear of producing fatal hemorrhage. It was not until 1928 that Wolf [7] first described the roentgen technic for diagnosis of esophageal varices. Kirklin and one of us (Moersch),[8] in 1931, called attention to the value of combining roentgenoscopic and esophagoscopic examination as a diagnostic measure and pointed out that it might also be of value in following the developments subsequent to treatment. With proper care, however, esophagoscopic procedures can be carried out with a minimum of risk.

NONSURGICAL TREATMENT

In addition to the surgical procedures to be described, many other methods have been advocated for the prevention and control of bleeding from esophageal varices. Measures which have been employed are dietary restrictions, the use of local applications to the varix (diathermy, local compression and so forth), injection of solutions into the general circulation to stimulate coagulation and venesection, as suggested by Drenckhahn,[9] to decrease the viscosity of the blood. Most of these procedures, however, are merely palliative and therefore of only temporary value.

SURGICAL PROCEDURES

"The spleen is an organ of contradiction and mystery; in health, of relatively unimportant function, in disease, a menace of grave import" (Mayo [10]).

Splenectomy.—The surgical procedure that has been employed most frequently is splenectomy. In discussing the surgical treatment of the spleen, Mayo,[11] in 1912, advised splenectomy in cases of splenic anemia on the theoretic ground that the enlarged spleen contributes to the reduction in "the number and value of the red blood corpuscles and low hemoglobin." Although this paper was read before the Clinical Congress of Surgeons of North America on Nov. 11, 1912, it was not published until 1913. He reported at that time that of the 27 splenectomies performed at the Mayo Clinic from April 6, 1904, to Oct. 21, 1912, 18 were for splenic anemia (5 patients had Banti's syndrome). There were 2

7. Wolf, G.: Die Erkennung von Ösophagus-Varizen im Röntgenbilde, Fortschr. a. d. Geb. d. Röntgenstrahlen **37**:890-893, 1928.

8. Kirklin, B. R., and Moersch, H. J.: Report of a Case of Roentgenologically Demonstrable Esophageal Varices Complicating Splenomegaly, Radiology **17**: 573-575 (Sept.) 1931.

9. Drenckhahn, C. H.: A New Approach to the Prevention of Hemorrhages from Esophageal Varices as Occur in Cirrhosis and Banti's Disease, Am. J. Digest. Dis. **6**:462-465 (Sept.) 1939.

10. Mayo, W. J.: The Enlarged Spleen, South. M. J. **21**:13-16 (Jan.) 1928.

11. Mayo, W. J.: Surgery of the Spleen, Surg., Gynec. & Obst. **16**:233-239 (March) 1913.

operative deaths in the 27 cases, both in cases of splenic anemia. The pathologic observations in the cases of splenic anemia showed chronic diffuse splenitis in 13 cases, endothelial proliferation (Gaucher's type) in 3 and lymphoid hyperplasia in 2 (lymphosarcoma in 1 and lymphoma [?] in 1). Twelve of the patients who had suffered from splenic anemia had been well for from one to seven years after operation. In 1929, Mayo [12] postulated that cirrhosis of the liver in cases of Banti's disease probably results from the toxic material gathered in the spleen and sent to the liver, which the liver, failing to detoxicate, attempts to encapsulate diffusely, with the introduction of general fibrosis. With the dysfunction of the enlarged spleen its already large blood supply greatly increases, and, since it is joined with the portal vein, it is estimated that the blood from the spleen supplies 20 per cent of the blood in the portal circulation. This is greatly increased with the increase in the size of the spleen, for the splenic vein and its branches are greatly increased in size. With removal of the spleen and ligation of the splenic vein this supply of blood to the portal vein would cease.

It was also assumed as a possibility that after the removal of the enlarged spleen in cases of splenic anemia the denuded parietal peritoneum of the posterior portion of the abdominal cavity, which had been in contact with the spleen, might be the site of formation of a network of collateral veins through which circulation between the portal system and the inferior vena cava could occur. In 1933, Kegaries [5b] showed that ligation of the branches of the splenic vein going to the cardia and the esophagus through the short gastric veins decreased the flow of blood through the esophageal veins when the spleen was removed and suggested that this is one of the important benefits of splenectomy. Splenectomy, however, did not completely solve the problem, for Mayo [12] in 1929, in his report of 148 cases of splenectomy for splenic anemia (with 14 deaths), stated that in about 10 per cent of the cases in which recovery took place after splenectomy the patients died at some time in the next ten years of hemorrhages from the stomach, probably from ruptured varices in the lower end of the esophagus. In 1931, Pemberton [13] reported the recurrence of hemorrhage from esophageal varices after splenectomy in approximately 50 per cent of 98 cases.

In a recent review of the results of various surgical procedures performed at the Mayo Clinic on patients with histories of bleeding esophageal varices, Giffin and two of us (Walters and McKinnon) [14] found

12. Mayo, W. J.: The Enlarged Spleen, California & West. Med. **30**:382-386 (June) 1929.

13. Pemberton, J. deJ.: Results of Splenectomy in Splenic Anaemia, Haemolytic Jaundice, and Haemorrhagic Purpura, Ann. Surg. **94**:755-765 (Oct.) 1931.

14. Giffin, H. S.; Walters, W., and McKinnon, D. A.: Unpublished data.

that 41 of 103 splenectomized patients had had no subsequent hemorrhages for periods varying from a few months to more than fifteen years after operation (table 1). It is to be noted that 80 of the patients underwent splenectomy alone; 12 underwent splenectomy and ligation of the coronary vein, with or without omentopexy, and 11 underwent omentopexy, but not ligation of the coronary vein, with the splenectomy.

From Jan. 1, 1908, to Dec. 31, 1934, inclusive, splenectomy was performed for splenic anemia in 180 cases at the Mayo Clinic. One

TABLE 1.—*Effect of Different Surgical Procedures on Bleeding Esophageal Varices in Cases of Splenic Anemia*

Procedure	Number of Cases	Bled Preoperatively	Bled Postoperatively	Untraced	Hospital Deaths	No Subsequent Hemorrhage
Splenectomy alone..	80	80	33	5	9	33
Splenectomy, ligation of coronary vein and omentopexy	3	3	2	0	0	1
Splenectomy and ligation of coronary vein	9	9	6	1	0	2
Splenectomy and omentopexy						
Preoperative bleeding	5	5	1	1	2	1
No preoperative bleeding	6	0	1	1	0	4

TABLE 2.—*Survival After Operation for Patients with Splenic Anemia Undergoing Splenectomy and Associated Surgical Procedures*

Five Year Survival After Operation *			Ten Year Survival After Operation *			Fifteen Year Survival After Operation *		
Total Patients	Traced Patients	Percentage of Survival	Total Patients	Traced Patients	Percentage of Survival	Total Patients	Traced Patients	Percentage of Survival
180	170	56.5	150	135	41.5	102	89	29.2

* The five year group comprises the patients on whom operation was performed five or more years prior to the time of inquiry, that is, on Dec. 31, 1934, or earlier; the ten year group comprises those on whom operation was performed on Dec. 31, 1929, or earlier; the fifteen year group comprises those on whom operation was performed on Dec. 31, 1924, or earlier.

hundred and seventy patients were traced, of whom 56.5 per cent lived for five years or more after operation. Of 150 patients on whom operation had been performed ten years or more before the time of inquiry, 135 were traced; of these, 41.5 per cent were living at the end of ten years, while 29.2 per cent lived for fifteen years or more after operation (table 2). When these results are compared with those in the operative cases in which the survival rate was based on the onset of symptoms (table 3) it is found that approximately 73 per cent lived five years or more; 56 per cent, ten years or more, and 52 per cent,

fifteen years or more. There was a hospital mortality rate in the 180 cases of 10 per cent. The causes of death (operative mortality) are shown in table 4.

Ligation of the Coronary Vein.—In 1929, Rowntree, McIndoe and one of us (Walters),[15] on the basis of anatomic studies of the portal circulation, advocated ligation of the coronary vein as a possible means of preventing bleeding from esophageal varices. The operation was

TABLE 3.—*Survival After Onset of Symptoms for Patients with Splenic Anemia Undergoing Splenectomy and Associated Surgical Procedures*

Five Year Survival After Onset of Symptoms *			Ten Year Survival After Onset of Symptoms *			Fifteen Year Survival After Onset of Symptoms *		
Total Patients	Traced Patients	Percentage of Survival	Total Patients	Traced Patients	Percentage of Survival	Total Patients	Traced Patients	Percentage of Survival
180	177	72.9	167	158	56.3	115	103	52.4

* The five year group comprises the patients whose symptoms began five or more years prior to the time of the inquiry, that is, on Dec. 31, 1934, or earlier; the ten year group comprises those whose symptoms began on Dec. 31, 1929, or earlier; the fifteen year group comprises those whose symptoms began on Dec. 31, 1924, or earlier.

TABLE 4.—*Causes of Death*

	Hospital Deaths
Mesenteric thrombosis (1 also bled from varices)	5
Pulmonary complications (2 pneumonia, 1 influenza, 1 pulmonary edema and 1 septic pleurisy)	5
Postoperative hemorrhage	3
Pulmonary emboli	2
Subdiaphragmatic and subhepatic abscesses	1
Hepatic insufficiency	1
Suicide, also had portal emboli	1

Eighteen deaths in 180 cases; hospital mortality, 10 per cent.

performed in 10 cases (table 5). Most of these were cases of cirrhosis of the liver.

Of the patients in these cases, 1 was operated on in 1929 and died in March 1932, having had repeated gastric hemorrhages. Of the 3 patients operated on in 1931, 2 were followed. One reported in 1935 that he had had two subsequent hemorrhages; the other died of cerebral hemorrhage two years after operation. Two patients operated on in 1933 died

15. Rowntree, L. G.; Walters, W., and McIndoe, A. H.: End Result of Tying of the Coronary Vein for Prevention of Hemorrhage from Esophageal Varices, Proc. Staff Meet., Mayo Clin. **4**:263-264 (Sept. 4) 1929. Walters, W.; Rowntree, L. G., and McIndoe, A. H.: Ligation of the Coronary Veins for Bleeding Esophageal Varices, ibid. **4**:146-147 (May 8) 1929.

of hepatic insufficiency. In the case of 1 there was a question of hemorrhage. In 1 of the 3 patients operated on in 1934, hepatic insufficiency with jaundice, ascites and edema developed in 1936. The second patient died in 1937 of hepatic insufficiency. This patient had had repeated gastrointestinal hemorrhages.

As was previously mentioned, ligation of the coronary vein and splenectomy were done in 9 cases (table 1). Those in which ligation of the coronary vein alone was done (4 cases) were cases of primary cirrhosis of the liver in which esophageal varices developed and in which hepatic insufficiency developed later and in most cases became the cause of death. Hence the true merits of this operation in the treatment of bleeding esophageal varices associated with splenic anemia cannot be definitely determined at this time, since ligation of the coronary vein

TABLE 5.—*Effect of Surgical Procedures on Bleeding Esophageal Varices in Patients with Cirrhosis of the Liver* *

Procedure	Number of Cases	Bled Preoperatively	Bled Postoperatively	Untraced	Died Subsequently	No Subsequent Hemorrhage
Ligation of coronary vein alone	4	4	2**	1	1, immediately 1, 2 years later	0
Ligation of coronary vein and omentopexy.	6	4	2**	3†	6, all but one died within 3 years	1

* One patient had syphilitic hepatitis.
** These patients have subsequently died, but we knew from follow-ups that they had bled before death.
† These patients are known to be dead, but their symptoms before death are unknown.

alone has been done on too few patients to justify any decision about its value.

Omentopexy.—Since many observers have noted from time to time that nature, in her desire to divert blood back into the general circulation around a cirrhotic liver, attaches the omentum to the abdominal wall, it was suggested by Talma and his associates [16] that an attempt be made to assist in the establishment of such a method of collateral circulation by placing the omentum in contact with the branches of the veins of the abdominal wall, particularly the superior epigastric vein and its branches. This could be done by scarifying the parietal peritoneum and suturing the omentum to it or by a modification which Mayo [17] described

16. Talma, Safe, Drummond, David, Morison and Rutherford, cited by Vander Veer, E. A.: Talma Operation for Cirrhosis of the Liver, with Report of Cases, Surg., Gynec. & Obst. **15**:278-281 (Sept.) 1912.

17. Mayo, W. J.: The Surgical Treatment of the Cirrhoses of the Liver and Their Complications, in Collected Papers of the Mayo Clinic, Philadelphia, W. B. Saunders Company, 1918, vol. 10, pp. 143-145.

in 1918 and which at that time he had used in 28 cases, namely, drawing the omentum up through the peritoneal incision and into the incised rectus muscle, suturing it in that position and closing the fascia carefully over it. In discussing the merits of this procedure he stated, "On several occasions we have gone down a second time in the vicinity of the previous operation, with a view of increasing the omental attachments, and have found such extensive compensatory circulation, almost entirely venous, that we were obliged to desist, and even with difficulty controlled the hemorrhage." In 1933, Pemberton,[18] in discussing the value of omentopexy as an adjunct to splenectomy performed on patients with splenic anemia, stated that he had used it in 15 cases, in which subsequent bleeding had not occurred. This must be qualified by a statement that he had not been in touch with some of the patients for a considerable period. In 1 of Pemberton's cases the sequence of events that followed omentopexy is worthy of description.

The patient in this case, who had Banti's disease, had complained bitterly of pain in the right side, and the clinician had suspected that the gallbladder or the appendix was the cause of this. It was felt, therefore, that it was advisable to explore the gallbladder and the appendix before performing splenectomy. At operation it was found that there was ascites grade 2 (on a basis of 1 to 4). This patient, according to the bromsulphalein test of hepatic function before operation, had had dye retention, grade 2, and at operation a biopsy specimen of the liver was taken, which showed portal cirrhosis, grade 1, and fatty liver, grade 1 plus. As the appendix and the gallbladder were not grossly diseased, the wound was closed after omentopexy had been performed. The patient's convalescence was uneventful; three weeks later the hepatic function test showed no dye retention, and she was allowed to go home. On her return to the clinic, six weeks after omentopexy, the hepatic function test again showed no dye retention. Splenectomy was done, and no free fluid was found in the abdomen. The under surface of the anterior abdominal wall, where the omentopexy had been previously done, was inspected, and the omentum running into the abdominal wall seemed to be particularly richly supplied with blood vessels. The patient went through the operation nicely and had a normal convalescence.

Likewise in 1933, Pemberton [18] suggested that if recurrent bleeding from esophageal varices occurred in patients on whom he had done omentopexy it would be a good idea to inject a sclerosing solution directly into the esophageal varices through the esophagoscope. In the performance of omentopexy Pemberton [18] expressed a preference for incorporating the omentum in the abdominal wall, lateral to the incision

18. Pemberton, J. deJ.: Personal communication to the authors.

for laparotomy, so as not to jeopardize healing from this incision. In describing his technic he stated:[13]

> After separation of the several layers of the abdominal wall for 3 centimetres from the edge of the wound, a small incision is made through the peritoneum and posterior sheath of the rectus abdominis muscle, and a segment of omentum 14 to 20 centimetres is then drawn up through this opening and sutured. Similar incisions are made in the muscle and anterior sheath of the rectus abdominis, at successive levels, each lower than the preceding one, 2.5 centimetres or more apart, and the omentum is drawn through these; the distal 5 to 8 centimetres is then buried beneath the skin.
>
> By bringing the omentum out in a steplike manner, conditions are established for the formation of new blood channels in each layer of the abdominal wall, and on account of the oblique course of the openings, the chances of troublesome herniation are minimized.

Injection of Sclerosing Solutions into the Esophageal Varices.—In 1933, one of us (Walters), in discussing Kegaries' paper,[5b] suggested that it might be advisable to inject a sclerosing solution into the varices around the esophagus at the time of ligation of the coronary vein. This procedure has recently been carried out by Grace.[19] At about this time, Pemberton and one of us (Moersch[20]) became interested in the possibility of injecting a sclerosing solution into the veins directly through an esophagoscope. Before attempting to carry out the procedure on human beings, the experimenters thought it would be advisable to perform it on experimental animals. An attempt was made to produce varices of the esophagus in dogs. While they could produce enlarged vessels over the thoracic wall of the animal, they were unable to produce enlargement of the veins in the esophagus. Consequently, the idea of employing the method on human beings remained in abeyance. During 1939, Crafoord and Frenckner,[21] of Stockholm, reported that they had carried out this form of therapy successfully on a human being and that the patient had remained free from symptoms for approximately three years after treatment. Their report encouraged one of us (Moersch[22]) to attempt to duplicate the procedure. The successful result obtained by this method was reported by him at a meeting of the American Association for Thoracic Surgery, and an abstract of the case follows (case 1).

---

19. Grace, E., in discussion on Moersch.[22]

20. Moersch, H. J.: Treatment of Esophageal Varices by Injection, Proc. Staff Meet., Mayo Clin. **15**:177-179 (March 20) 1940.

21. Crafoord, C., and Frenckner, P.: New Surgical Treatment of Varicose Veins of the Oesophagus, Acta oto-laryng. **27**:422-429, 1939.

22. Moersch, H. J.: The Treatment of Esophageal Varices by Injection of a Sclerosing Solution, read at the Meeting of the American Association for Thoracic Surgery, Cleveland, June 6-8, 1940.

CASE 1.—A man 30 years of age came to the Mayo Clinic in February 1940 with a history of recurrent gastrointestinal hemorrhage. A diagnosis of Banti's disease or cirrhosis of the liver had been made elsewhere. The patient apparently had enjoyed the best of health until 1928, in which year he was suddenly stricken with a severe attack of hematemesis. He was hospitalized, and repeated transfusions of blood were necessary. The patient recovered from this attack satisfactorily and remained well for approximately one year, at the end of which he suffered a similar attack and transfusions of blood again were required. At this time enlargement of the spleen was noted and splenectomy was advised. At operation the spleen, which was approximately nine times the normal size, was removed. There is no record of the exact character of the spleen. After operation the patient did not experience recurrence of bleeding until 1936, in which year he again began to suffer from hemorrhage. Since that time he had been having recurrent attacks of bleeding which occurred with increasing frequency, and during the year prior to his coming to the clinic he had had four attacks, the last one occurring in December 1939.

On physical examination nothing of unusual consequence was found. The significant observations at laboratory investigation were as follows: There was a reduction in the value for hemoglobin to 9.6 Gm. per hundred cubic centimeters,

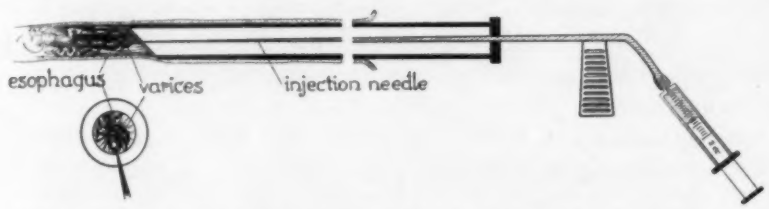


Fig. 4.—Instrument and needle employed in the injection of sclerosing solutions into esophageal varices. (From Moersch, H. J.: The Treatment of Esophageal Varices by Injection, Proc. Staff Meet., Mayo Clin. **15**:177-179 (March 20) 1940.

and the blood smear was reported as showing macrocytic hypochromic anemia of the type associated with cirrhosis of the liver. Roentgen examination of the esophagus revealed esophageal varices involving the lower third of the gullet. It is rather interesting that the test for hepatic function did not show retention of dye.

A clinical diagnosis of cirrhosis of the liver with associated esophageal varices was made. One of us (Walters), who saw the patient in surgical consultation, suggested that this might be a suitable case in which to attempt control of the bleeding by injection of a sclerosing solution into the varices. In spite of the risk involved, the patient was rather anxious to have us attempt the procedure, because he lived in constant fear of a fatal hemorrhage.

Esophagoscopic investigation was made with local anesthesia, and markedly enlarged varices involving the lower third of the esophagus were readily found. They were of such size that they almost completely obstructed the lumen of the gullet. They formed large, bluish nodular prominences which were soft and compressible. A suitable vein was selected for injection, and with a 25 caliber needle (fig. 4) 0.5 cc. of a solution of 2.5 per cent sodium morrhuate was injected. Much to the surprise of one of us (Moersch), the procedure was accomplished with practically no bleeding. Injections were given three more times at four day intervals, 1 cc. of the solution being injected on each of these occasions.

The change that took place after the injection treatment was striking. The vessels into which the sclerosing solution had been injected lost their bluish color and became yellowish gray; they were firmer and smaller, and the surrounding mucous membrane became less congested. After the last injection the lumen of the esophagus, which had been almost completely obstructed by the enlarged varices, returned almost to its normal caliber, and it was possible to look into the stomach without difficulty.

In a recent communication received from the patient, he stated that he has had no further bleeding since his treatment, that the value for hemoglobin has increased to 81.9 per cent and that he has been feeling better than he has for several years.

Sufficient time has not elapsed in which to determine the efficacy of the injection treatment of esophageal varices, and it may be found necessary to repeat the procedure if further bleeding should take place. It is still uncertain whether the injection should precede or follow splenectomy and ligation of the coronary vein. At present one of us (Moersch) is inclined to believe that it would be advisable to carry out the splenectomy before the injection is attempted. It seems, however, that this procedure is worthy of serious consideration, as it offers a possible method of controlling bleeding that has not responded to any other form of therapy and thus may be successful in prolonging the life of the patient.

Since this report, one of us (Moersch) has carried out or attempted injection treatment of esophageal varices in 5 additional cases.

Case 2.—The patient was a man aged 40 years who had syphilitic hepatitis. He had been having gastric hemorrhages. Roentgen examination was reported as showing varices involving the lower half of the esophagus. The spleen was found to be enlarged, as was the liver. On esophagoscopic examination the varices were found to involve the lower part of the esophagus. Although they were numerous, they were very small in caliber, and Moersch had a great deal of difficulty in trying to insert the needle into the veins. Because of this, he did not feel that further injection was advisable at this time.

Case 3.—The patient was a woman 52 years of age. In October 1939 she had her first massive gastrointestinal hemorrhage. She had no further trouble until eight days before admission to the clinic, on June 8, 1940. She stated that cholecystectomy had been performed seven years previously. On physical examination the spleen was not palpable. The patient was markedly anemic. One of us (Walters) saw her in surgical consultation and did not feel that splenectomy was advisable. Roentgen examination showed varices of the lower third of the esophagus. On esophagoscopic examination, large veins were found involving the lower half of the esophagus. Sodium morrhuate was injected into these and the patient was permitted to go home, with instructions to return in three months for further examination. There was no retention of dye, and there has been no further bleeding.

Case 4.—The patient was a man aged 20 years. At 2 years of age he had severe malaria, and since then he had had an enlarged spleen. At the age of 8

years a diagnosis of splenic anemia was made and splenectomy was performed. One year after splenectomy the patient had his first gastrointestinal hemorrhage. Since then he had had repeated severe gastrointestinal hemorrhages, with loss of blood both by hematemesis and by bowel. Transfusions had been required frequently. In January 1939 ligation of the coronary vein was carried out, without marked benefit. The hepatic function test gave normal results. His blood volume had been kept low on purpose, in an attempt to prevent bleeding, but this had failed. Roentgen examination made here showed extensive varices involving the greater portion of the esophagus. Esophagoscopic examination likewise showed extensive varices involving most of the gullet. Repeated injections were carried out, and the patient was permitted to go home, with instructions to return for further examination in three months. He has had no further bleeding since the treatment.

Case 5.—A man aged 46 years gave a history of recurrent attacks of hematemesis and melena since Sept. 25, 1938. He had had severe hemorrhages as frequently as every two weeks. Repeated blood transfusions had been required. The patient first came to the clinic in March 1939, and a diagnosis of esophageal varices was made. The spleen was not palpable at this time. Roentgen examination revealed esophageal varices involving the greater portion of the esophagus. Esophagoscopic study revealed large varices involving the lower half of the esophagus and the cardiac portion of the stomach. The patient was seen in surgical consultation, but operation was not advised. He continued to have bleeding and returned to the clinic in June 1940, at which time the value for hemoglobin was 48 per cent and the erythrocyte count 4,800,000 per cubic millimeter of blood. A test of hepatic function showed no retention of dye. When the first injection was attempted, the patient had a very severe hemorrhage and it was necessary to pack the esophagus. This controlled the bleeding nicely, and the patient recovered well. Further injections were carried out. While still under treatment he had a very severe hemorrhage, and at present he is in the hospital receiving transfusions. During the hemorrhage the value for hemoglobin was reduced to 9 per cent. Further injections are to be carried out because of his insistence and because of the fact that there is practically no alternative in his case.

Case 6.—The patient was a man aged 37 years. He first began to have melena and hematemesis in 1934. It is rather interesting that gastroscopic examination was made six times elsewhere in an effort to locate the cause of the bleeding, but no bleeding point was found in the stomach, and no bleeding was produced as a result of the procedures. He first came to the clinic in 1934, when a diagnosis was made of hepatic cirrhosis with associated varices of the lower end of the esophagus. He has had repeated bleeding since that time, and repeated transfusions have been required. An interesting feature in his case is that he has had dye retention of grade 3 but that recently this has been only of grade 1. It has been only during the past year that the spleen has become slightly palpable. Roentgen examination showed varices involving the lower end of the esophagus. Esophagoscopic examination, however, showed the varices to run up into the upper third of the gullet. Injections have been given, with marked decrease in the size of the varices. The patient recently has had one very small episode of bleeding, and esophagoscopic examination since that time has revealed that a small vessel in the middle third of the esophagus had apparently leaked. Further injection of the sclerosing solution into this vessel has been carried out, and the patient has been getting along nicely.

We think that it must be emphasized that sufficient time has not elapsed to enable us to arrive at any definite conclusions as to the efficacy of this form of treatment. It may be pointed out that roentgen examination of the esophagus does not always demonstrate the varices. This is illustrated by a patient on whom one of us (Walters) recently performed splenectomy. In her case roentgen examination of the esophagus gave negative results, but on esophagoscopic examination we found large varices involving the lower third of the esophagus and almost occluding its lumen.[23] It is also of interest that the esophageal varices generally have been found to be much more extensive than the roentgenogram would lead one to believe. In practically all of the cases the varices were found to extend up into the upper third of the esophagus. It may also be pointed out that instrumentation can apparently be carried out without a great deal of danger of producing hemorrhage. This would seem to emphasize that trauma from the passage of food through the gullet is probably of only minor importance in the production of bleeding.

SUMMARY

Esophageal varices develop as a result of obstruction of the portal and splenic veins, and bleeding occurs because of their superficial position in relation to the esophageal mucosa. Although Banti's disease and splenic anemia are the conditions most frequently associated with esophageal varices, the former cannot be regarded as distinct clinical entities.

The surgical treatment of splenic anemia has been directed toward removal of the enlarged spleen on the assumption that it was contributing toward the destruction of red blood cells and, in addition, because splenectomy reduces the amount of blood flow to the portal vein by an amount ranging from a normal percentage of approximately 20 to a much larger percentage when the splenic vein and its branches enlarge with the enlargement of the spleen. In addition, in splenectomy the veins communicating between the splenic vein and the cardia and esophagus through the short gastric veins and gastrolienal ligament are interrupted by division and ligation, which assists in decreasing the amount of venous blood coursing through the esophageal varices. After removal of the spleen the denuded surface of parietal peritoneum forming its previous bed may be the site of formation of collateral veins between the portal and the caval system.

Although splenectomy performed for splenic anemia has been followed by good results in a large series of cases, it is apparent that

23. Although the spleen was about four times the normal size (weight, 910 Gm.) in this case, with a considerable degree of splenitis, with fibrosis of the pulp and with marked phlebitis of the splenic vein and its branches, the liver appeared grossly to be normal.

this operation, even when combined with ligation of the coronary vein or with omentopexy, does not prevent recurrence of bleeding from the esophageal varices in more than 38 per cent of the cases. This is in keeping with the earlier reports of Mayo,[12] who said that "in about 10 per cent of the cases in which recovery has taken place after splenectomy, the patients have died some time in the next ten years of hemorrhage from the stomach, probably from ruptured varices in the lower end of the esophagus."

Although it has been shown that in the presence of portal cirrhosis a considerable flow of blood occurs through the coronary vein, which anastomoses with the internal mammary vein at the cardiac end of the stomach and in the esophagus and that ligation of this vein in the gastrohepatic omentum will serve to interrupt this flow of blood, this procedure has been used alone in too few cases of splenic anemia to justify any conclusion relative to its merits in reducing the incidence of hemorrhages from esophageal varices in such cases.

The recent successful obliteration of esophageal varices by the injection of sclerosing solutions into them through the esophagoscope and the report of 6 cases in which the injection was done at the Mayo Clinic give indication that this is a procedure worthy of trial to determine the permanence of its efficacy in causing obliteration of esophageal varicosities.

ESOPHAGEAL DIVERTICULA

FRANK H. LAHEY, M.D.
BOSTON

HISTORICAL DATA

In 1764 Ludlow [1] observed "preternatural pockets" in the esophagus at autopsy.

In 1816 Bell [2] published in his volume of "Surgical Observations" a paper entitled "A Praeternatural Bag, Formed by the Membrane of the Pharynx."

CLASSIFICATION

In 1877, Zenker and von Ziemssen [3] were the first to classify diverticula of the esophagus into traction and pulsion types. They also made a study of the literature (23 cases) and added 5 of their own, in which autopsy was performed.

In 1922, Bensaude, Grégoire and Guénaux [4] made a new classification of esophageal diverticula—those of the esophagus are distinguished from pharyngoesophageal diverticula.

Traction Diverticula.—In 1840, Rokitansky [5] first correctly described traction diverticula.

Etiology.—In 1816, Bell [2] described the condition as due to distention of the pharynx from ineffectual attempts to swallow, with resultant protrusion of the inner coats of the pharynx through hypertrophied bundles of the inferior constrictor pharyngis muscle.

In 1921, Kulenkampff [6] said that the condition is analogous to inguinal hernia and is produced by establishment of a sac in the site of a congenital muscular hiatus which is covered in with elastic tissue.

---

1. Ludlow, A.: Obstructed Deglutition, from a Preternatural Dilatation of, and Bag Formed in, the Pharynx, M. Obs. Soc. Physicians London **3**:85-101, 1762-1767.

2. Bell, C.: Surgical Observations, London, Longman [and others], 1816, pp. 64-70.

3. Zenker, F. A., and von Ziemssen, H.: Krankheiten des Oesophagus, in von Ziemssen, H.: Handbuch der speciellen Pathologie und Therapie, Leipzig, F. C. Vogel, 1877, vol. 7 (supp.), pp. 50-87.

4. Bensaude, R.; Grégoire, R., and Guénaux, G.: Diagnostique et traitement des diverticules oesophagiens, Arch. d. mal. de l'app. digest. **12**:145-203, 1922.

5. Rokitansky, C.: Spindelförmige Erweiterung der Speiseröhre, Med. Jahrb. d. k. k. österr. Staates **21**:219-222, 1840.

6. Kulenkampff, D.: Zur Aetiologie, Diagnose und Therapie der sogenannten Pulsionsdivertikel der Speiseröhre, Beitr. z. klin. Chir. **124**:487-515, 1921.

Symptoms.—In 1900, Starck [7] divided symptoms into prodromal, direct and indirect.

Surgical Treatment.—In 1830, Bell proposed establishment of a fistula to empty the diverticulum of its contents; this was practiced in 1877 by Nicoladoni [8] in Vienna, Austria.

In 1850, Kluge conceived the idea of excision, which was first done in 1884 by Niehans [9] on a patient with both goiter and esophageal diverticulum.

In 1892, von Bergmann [10] reported a case of successful extirpation, and in the same year Kocher [11] performed the operation, with healing by first intention.

In 1896, Girard [12] suggested a new method—invagination of the diverticulum and suturing of the esophageal walls.

In 1917, Bevan [13] elaborated this procedure. He inverted the outer half of the sac and buried the stump with a series of purse string sutures.

In 1912, Schmid [14] proposed diverticulopexy, which was performed in 1917 by Hill [15] on a patient, with temporarily good results.

From 1910 to 1915, the popularity of surgical treatment was inhibited by the high mortality associated with one stage procedures.

In 1909, Goldmann [16] performed two stage operations; the pouch was freed, its pedicle ligated with silk, the wound packed and the sac fixed to the surface of the wound. This procedure resulted in sloughing of the diverticulum, and on the eighth day a fistula formed, which healed in two months.

---

7. Starck, H.: Die Divertikel der Speiseröhre, Leipzig, F. C. W. Vogel, 1900.

8. Nicoladoni, K.: Behandlung der Oesophagusdivertikel, Wien. med. Wchnschr. **27**:606-607, 1877.

9. Niehans, cited by Zesas, G.: Beitrag zur chirurgischen Behandlung des Speisenröhren-Divertikels, Deutsche Ztschr. f. Chir. **82**:577, 1906.

10. von Bergmann, E.: Ueber den Oesophagusdivertikel und seine Behandlung, Arch. f. klin. Chir. **43**:1-30, 1892.

11. Kocher, T.: Das Oesophagusdivertikel und dessen Behandlung, Cor.-Bl. f. schweiz. Aerzte **22**:233-244, 1892.

12. Girard, C.: Du traitement des diverticules de l'œsophage, Assoc. franç. de chir. Proc. verb. **10**:392-407, 1896.

13. Bevan, A. D.: Pulsion Diverticulum of Esophagus, S. Clin. Chicago **1**:449-457, 1917.

14. Schmid, H. H.: Vorschlag eines einfachen Operations-verfahrens zur Behandlung des Oesophagus-divertikels, Wien. klin. Wchnschr. **25**:487-488, 1912.

15. Hill, W.: Pharyngeal Pouch Treated by Diverticulopexy, Proc. Roy. Soc. Med. (Sect. Laryng.) **11**:60, 1917-1918.

16. Goldmann, E. E.: Die zweizeitige Operation von Pulsiondivertikeln der Speiseröhre, Beitr. z. klin. Chir. **61**:741-749, 1909.

In 1916, Murphy [17] improved this method by implanting the sac in the wound with its neck unligated, thus preventing sloughing, and resected it two weeks later.

In 1918, Judd [18] reported a modification of this method; the edges of skin were sutured to the neck of the sac; the wound was closed, and

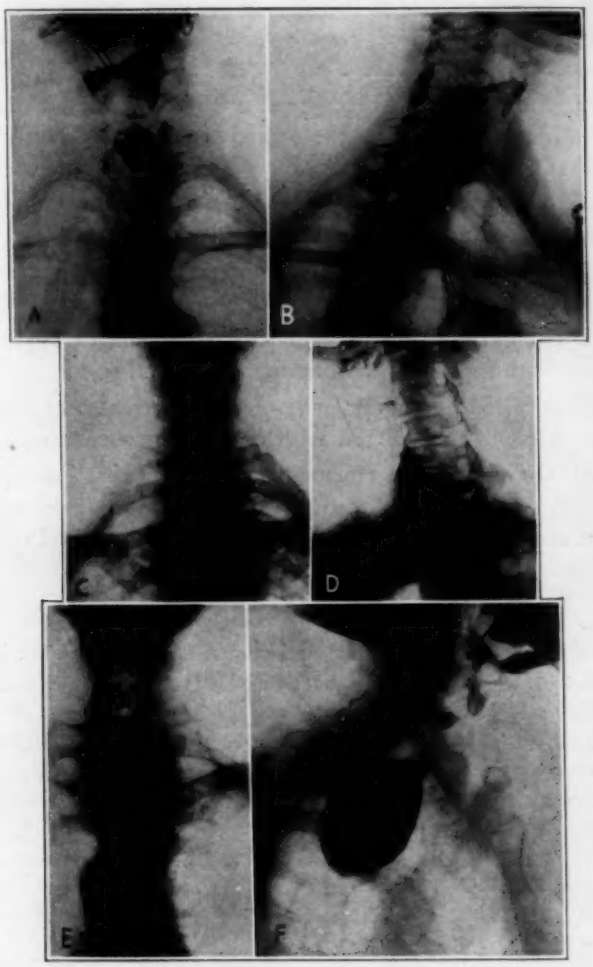


Fig. 1.—*A* and *B*, examples of the small type of pharyngoesophageal pulsion diverticulum. *C* and *D*, examples of the intermediate type. *E* and *F*, examples of the large type.

17. Murphy, J. B.: Diverticulum of Esophagus: Conservative Treatment, S. Clin. Chicago **5**:391-395, 1916.

18. Judd, E. S.: Esophageal Diverticula, in Collected Papers of the Mayo Clinic, Philadelphia, W. B. Saunders Company, 1918, vol. 10, p. 15.

the sac was left as in an unopened colostomy stoma, lying on the skin. Ten to twelve days later, when healing had taken place within the wound and about the neck of the sac, it was cut away and its edges turned into the esophagus.

In 1933, I [19] modified the two stage operation. In small sacs the tip of the sac after dissection was sutured to the sternohyoid muscle at

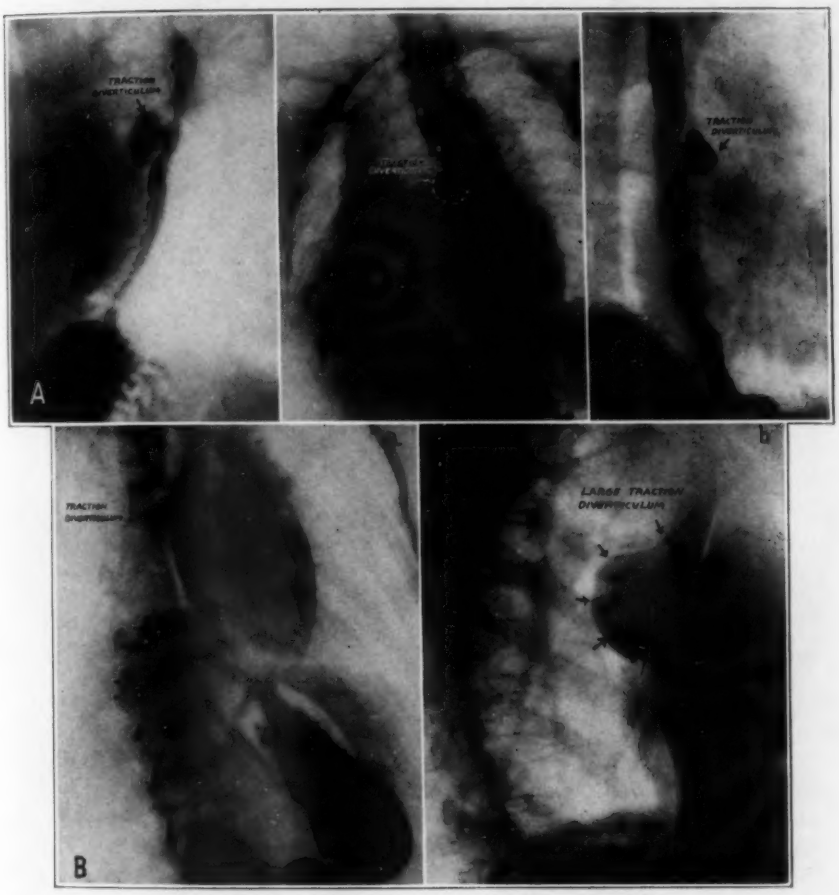


Fig. 2.—*A*, three examples of traction diverticula. Note the tendency of these diverticula to be pulled out laterally and not in a dependent position. *B*, further types of traction diverticula. Roentgenogram *a* represents the laterally pulled diverticulum, and roentgenogram *b*, the largest and most dependent type of traction diverticulum ever seen at this clinic. All of these have been handled satisfactorily by dilation. Note in roentgenogram *b* that, even though the diverticulum is of considerable size, the size of its opening into the esophagus is nearly half that of the complete diameter of the diverticulum, thus making it easily possible to empty itself.

19. Lahey, F. H.: Esophageal Diverticulum, J. A. M. A. **101**:994-997 (Sept. 23) 1933.

a level higher than its neck within the wound, and in large diverticula the sac was implanted in the wound, pointing upward. This prevented food from entering the wound between stages and caused the fistula which occurred after the second stage to close early.

Esophageal diverticula divide themselves into two main types, true diverticula, represented by the traction type of sac made up of all the

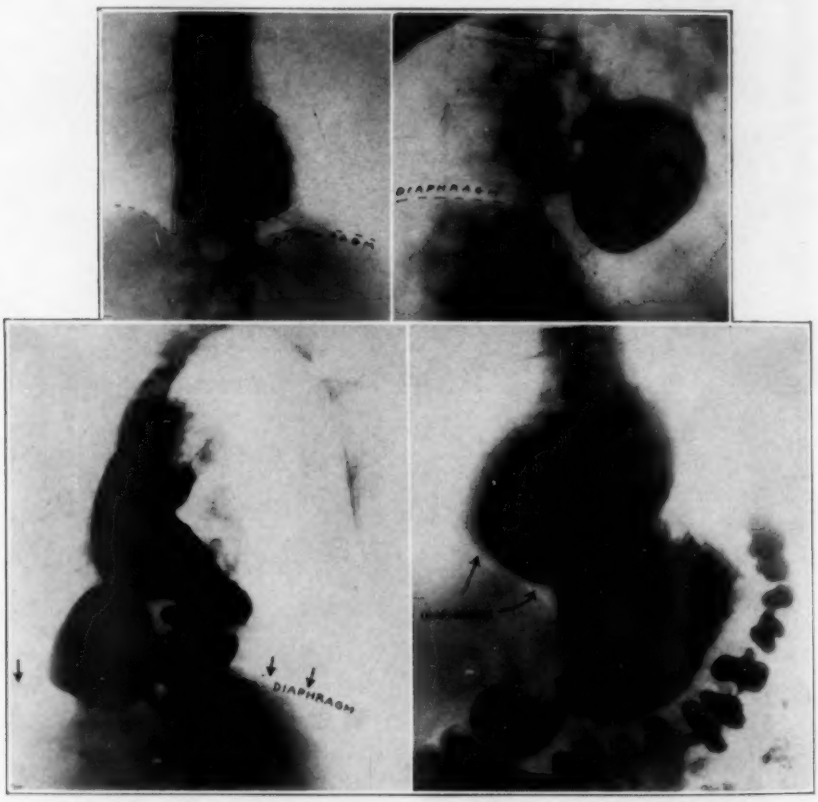


Fig. 3.—Supradiaphragmatic pulsion diverticula. The chief complaint in cases of this type of diverticulum is regurgitation of the foul contents of the sac, bringing about nausea together with regurgitation of the contents of the sac into the throat during sleep and thus waking the patient.

coats of the esophagus, and false diverticula, represented by the pulsion type so commonly seen at the pharyngoesophageal junction and so named the pharyngoesophageal diverticulum.

Esophageal diverticula occur at three levels: those at the pharyngo-esophageal junction, represented by the pharyngoesophageal pulsion diverticulum (fig. 1); those in the intrapleural portion of the esophagus,

particularly at the level of the main bronchial stem, represented by the traction diverticulum (fig. 2) and those just above the diaphragm, the pulsion type (fig. 3). By far the most common type and certainly the type most prone to produce troublesome symptoms is the pharyngoesophageal diverticulum.

PHARYNGOESOPHAGEAL DIVERTICULUM

The pharyngoesophageal diverticulum has been well described as corresponding in its origin to an inguinal hernia. It is a true herniation of mucosa and submucosa through the lowest fibers of the inferior constrictor muscle as they run transversely or through the obliquely dividing fibers of the cricopharyngei muscles on the posterior aspect of the esophagus (fig. 4). As the cricopharyngei muscles spread off from the last fibers of the inferior constrictor muscle to become longitudinal and envelop the esophagus, they leave on the posterior wall of the esophagus a weak point, bounded above by the most inferior fibers of the lowest constrictor muscles and laterally by the obliquely descending fibers of the cricopharyngei muscles. This leaves a point or dimple

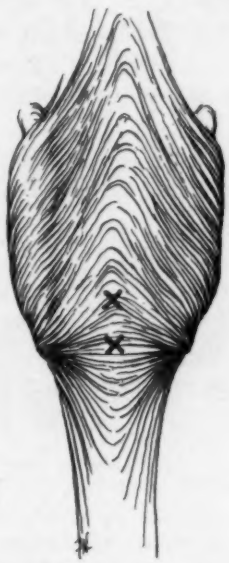


Fig. 4.—Diagrammatic illustration of the posterior aspect of the junction of the pharynx and the esophagus. The two points marked *x* represent the two levels at which pulsion diverticula herniate through—the upper *x* is in the lowest fibers of the inferior constrictor muscle, and the lower *x* is at the pharyngeal dimple, the defect in the posterior wall where the cricopharyngei muscles diverge from the lowest fibers of the inferior constrictor muscle.

on the posterior wall of the pharynx at the cricopharyngeal junction unsupported or weakly supported by muscular covering.

It has been suggested, owing to an incoordination between the impulses of the inferior constrictor muscles and the cricopharyngei muscles, that material as it is propelled downward by the constrictors is resisted in its descent by any uncoordinated action of the cricopharyngei muscles, thus tending to produce a bulging through this weak point.

It is probable that in some persons there is at this point, as is true with inguinal hernia, a congenital weakness in muscular covering. Taken together with neuromuscular incoordination, this results in bulging of the mucosa and submucosa at this weak point, just as such bulging occurs through a weak inguinal ring in the early stages of an inguinal hernia. At this period the first stage of the pharyngoesophageal diverticulum occurs, diagrammatically shown in insert 1 in figure 5 *a* and, as

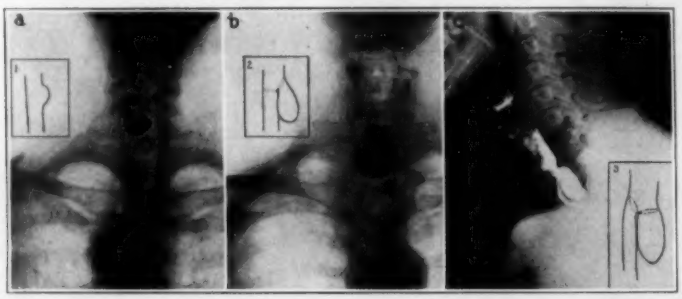


Fig. 5.—Roentgenogram *a* demonstrates the smallest type of esophageal diverticulum in its very earliest stage. Note that at this stage the sac has no neck. In insert *1* may be seen diagrammatically the early stage, in which the submucosa bulges through the fibers of the inferior constrictor muscle without as yet having produced a sac possessing a neck. Roentgenogram *b* represents a fully developed esophageal pulsion diverticulum with a definite neck; it is now at an operable stage. Note (insert *2*) the diagrammatic illustration of the relation of the false opening into the diverticulum to the true opening into the esophagus. Note that the opening into the esophagus is in the lateral position at this stage, so that food still passes readily through the transverse opening into the true esophagus without difficulty. Roentgenogram *c* represents the stage of development in the esophageal diverticulum in which, owing to traction on the sac, the opening into the diverticulum (insert *3*) is transverse and the opening into the esophagus lateral. One can appreciate from this diagram how food passes more readily into the diverticulum sac than into the esophagus and how traction with swallowing on the food-filled sac pulls the lips of the laterally placed opening into the esophagus together, which interferes further with the entrance of food into the true esophagus.

seen in an actual case, shown in the roentgenogram (fig. 5 *a*). At this period of development of the diverticulum no semblance of a sac is present; only a teatlike projection of mucosa bulges between the oblique

fibers of the cricopharyngei muscles and the almost transverse fibers of the inferior constrictor muscle.

At this period of development appears the earliest symptom associated with esophageal diverticula, solely the feeling of a piece of food stuck in the throat, causing an annoying cough in an endeavor to get it up. This is undoubtedly due to crusts of bread, flakes of cereal or any other dry bits of food lodging within the teatlike projection of mucosa, producing the same disagreeable discomfort as may be produced by a particle of food remaining persistently lodged in any portion of the pharynx.

As at this stage there is no neck to the sac, no operation should be considered. In my early experience with diverticula I have explored such diverticula at this stage and have not been able to undertake satisfactory operative repair. Since patients with diverticula at this time have no obstructive symptoms, no symptoms of regurgitation and only those symptoms occasionally brought about by particles of food lodging in the sac, there is no immediate necessity for operation, and one can with safety wait for further development of the sac. Wide and frequent dilation of the diverticulum at this period delays the development of the sac by dilating the fibers of the cricopharyngei muscles at the lower part of the neck of the sac. In the experience of this clinic this does not, however, prevent the ultimate occurrence of a progressively enlarging diverticulum with a well developed sac and neck.

In figure 5 *b* may be seen the completely formed diverticulum sac with a well developed neck and a dependent sac. This stage represents further advance from the first, or teatlike, stage and complete herniation of the pharyngoesophageal mucosa and submucosa between the most inferior fibers of the lowest constrictor and the oblique fibers of the cricopharyngei muscles. It represents the development of a true hernial sac, comparable with the sac of a fully developed oblique inguinal hernia.

In this second stage of the development of a pulsion pharyngoesophageal diverticulum it is to be noted (fig. 5 *b*) that while the fully developed sac is still moderately small the opening into the sac is in the oblique direction on the lateral wall of the esophagus, and the opening into the true esophagus is still in the transverse position. This is of importance because at this stage a large portion of the food still passes satisfactorily by the lateral opening of the esophagus into the diverticulum and descends along the longitudinal esophagus into the stomach without obstructive symptoms.

As this stage the only inconveniences suffered by the patient are those related to the accumulation of food and mucus within the sac. There is frequently regurgitation of food eaten at a previous meal, mixed with mucus. At this stage the patients frequently complain that as they swallow food gurgling noises may be heard in the throat, due to the movement of the ascending and descending sac agitating the air mixed with

fluid and food within the sac. Patients at this stage not infrequently present themselves for operation because, they state, as a result of these noises they are objects of curiosity on the part of those who have meals with them. They frequently complain that their companions at meals are curious as to what causes the noises in the throat when they swallow.

Stage 3, illustrated in figure 5 *b*, and insert 3, represents the most advanced stages of a pharyngoesophageal diverticulum. At this stage the sac has become large and frequently has descended into the mediastinum. Downward traction on the food-filled sac converts the sac opening (fig. 5 *c*, roentgenogram and insert) into a transverse one. Downward traction on the sac so angulates the esophagus that the direct course of descent of food is into the diverticulum itself; this tends not only to enlarge the sac but to force it by the weight of its retained food and by the traction on the food-filled sac with swallowing always in a downward direction into the mediastinum. As the large sac descends into the mediastinum and angulates the esophagus, it likewise angulates the true opening into the esophagus, so that it assumes a lateral position due to traction of the food-filled sac on the lateral esophageal wall. This true opening into the esophagus tends to have its lips so pulled together that, as it is viewed through the esophagus, the opening into the true esophagus is frequently represented only by a longitudinal slit. This accounts for the often encountered difficulty of passing an esophagoscope by the sac into the true esophagus. It is at this stage that obstructive symptoms tend to appear, since the first food swallowed fills the sac, makes traction on the now laterally placed true opening to the esophagus and tends to close it. Food then finds its way with difficulty through this opening into the true esophagus and thence into the stomach.

When I first began to deal with esophageal diverticula, because the food-distended sac pressed on the lateral wall of the esophagus it was my impression that the obstructive symptoms related to esophageal diverticula were largely caused by this mechanical pressure. I was unaware of the real cause of obstruction until esophagoscopic study of a number of patients demonstrated that the opening into the diverticulum itself was a true transverse one and that the opening into the true esophagus was laterally placed, with its lips so pulled together that food could not pass into it.

In the early stages of esophageal diverticula the symptoms are of insufficient magnitude to cause the patients seriously to consider seeking operative intervention. Many diverticula on which my associates and I have operated have undoubtedly existed for a number of years without their presence having been discovered either by the patient or by the physician.

One of the reasons that the patients come to operation is that they have been made unhappy by regurgitation of previously eaten food

from the diverticulum sac, not infrequently on awkward and unexpected occasions. They have requested operation, as has been stated, not infrequently because they have been annoyed by the attention directed to them as they ate as the result of the gurgling within the sac due to air mixed with fluid as the sac ascends and descends with deglutition. They have also not infrequently sought surgical relief because the fluid-filled sac has spilled over at night when they were asleep, the fluid contents of the sac trickling down into the larynx and awakening them with violent coughing. As the result of this experience, 2 patients have inhaled such amounts of material spilled from the sac that abscesses of the lung have resulted, requiring preliminary drainage before operation on the esophageal diverticulum could be carried out. A large number of patients have, of course, sought operation because they have reached the third stage of the development of an esophageal diverticulum, with complete obstruction or ability to get little, if any, food into the stomach.

It is of interest to state, I believe, that fewer and fewer patients with very large esophageal diverticula are coming for operation, because physicians are becoming more familiar with the diagnosis of this condition, are more aware of the danger of delay in treating it and have developed greater confidence in the safety of its operative correction. The patients are now being sent for operation at much earlier stages, while the sacs are reasonably small.

A few patients have come to operation with complicated situations. Three have been operated on whose diverticulum sacs had been perforated elsewhere, before they came to this clinic, by either an esophagoscope or an esophageal dilator, serious mediastinitis resulting. One required posterior mediastinotomy, and 2 required gastrostomy and drainage of the mediastinum anteriorly before they recovered from the resulting mediastinitis. In spite of the fact that one would expect a great many adhesions in the mediastinum and about the neck of the sac to follow such an inflammatory reaction, this was not the case, and dissection of the sac was accomplished in these patients with little more added difficulty than in those cases in which no previous mediastinal infection and infection of the fascial plane had taken place. None of these patients, however, were operated on earlier than several months after recovery from the mediastinitis and cervical cellulitis.

One patient had a carcinoma of the esophagus limited to the body of the diverticulum sac (fig. 6). Resection of the sac by the two stage method, together with a considerable portion of the adjacent esophagus, resulted in a most satisfactory outcome. The case is, however, of too recent origin for one to make any statement as to nonrecurrence of the carcinoma. The wound has healed completely; the esophagus has been dilated, and the patient is swallowing well. It was necessary in this case,

because of the amount of esophagus which was removed together with the sac and its neck, to feed the patient through a Levine tube inserted into the stomach through the nose.

It has frequently been advised that patients with complete obstruction resulting from large mediastinal diverticulum sacs be submitted to preliminary gastrostomy. This is undoubtedly a wise procedure in cases in which obstruction to the esophagus has been present for a considerable

Fig. 6.—Roentgenogram demonstrating the occurrence of carcinoma arising in the mucosa of the lining of the diverticulum sac and limited to the diverticulum. Not only was the diverticulum sac with its contained carcinoma removed in this case, but in order to obtain a wide margin of safety a considerable portion of the wall of the true esophagus was also removed.

period, with marked loss of weight, dehydration and general inanition. It is not necessary, however, in cases in which there is only incomplete interference with passage of food through the esophagus into the stomach. I have suggested and practiced (fig. 9) the plan of completely dissecting the large sacs, implanting them in the wound and then inserting a feeding tube through a purse string suture in the dome of the sac. With

the tube sutured into the sac it may be guided down the esophagus into the stomach, and feeding may be immediately begun without the added complication of a gastrostomy.

One can, of course, introduce a nasal catheter while the cervical wound is open, guide it past the sac into the esophagus after the sac has been freed and feed the patient in this way. It is far more comfortable for the patient to have the feeding tube introduced through the dome of the sac and into the esophagus, resulting in less irritation and discomfort than is the case when a nasal tube is left in the esophagus for a number of days.

The type of anesthesia employed in the operative treatment of esophageal diverticula is of considerable importance. My associates and I have employed regional and cervical block anesthesia in nearly all of these cases, because with the spilling of sac contents with operative handling of the sac it is so important for the patients to retain their throat reflexes to prevent pulmonary infections due to aspiration of these contents. In addition, this type of anesthesia is desirable because, with the patient conscious, he can be asked to swallow and thus make the sac ascend and descend, so that it can easily be visualized and dissected from below anteriorly, from above and along its opposite posterior wall.

There has been a good deal of debate at medical meetings and in the literature, as with so many other surgical procedures, as to whether one should employ the one stage operation in which the sac is completely dissected and its neck transfixed, ligated or sutured or the two stage procedure about which I have frequently written, in which at the first stage the sac is completely dissected, implanted high in the wound or in the skin and removed at the end of eight to ten days, when the fascial planes and mediastinum have been walled off.

There will always be differences of opinion regarding one stage and two stage operative procedures. They will be settled by a variety of circumstances: the extent of experience of the surgeon dealing with these cases; the type of cases with which he has to deal; the individual surgeon's ability and equipment to handle such cases, and the temperament of the person responsible for the patient, in terms of caution and responsibility.

I have repeatedly stated that I feel sure that the one stage procedure can be done with reasonable safety but that in a large series of cases the mortality rate, however small it may be in both groups, will be somewhat more favorable in the group done in two stages. It has always been my feeling that I am less interested in saving the patient's time than I am in saving the life of that last patient that makes up even a very small mortality percentage. I have now operated on 118 patients for esophageal diverticula. There has been but 1 fatality in the entire series, and that

in a man of 81. This patient died not of the operation for his diverticulum but of uremia. Nevertheless, I am convinced that mortality as related to operative procedures cannot be explained away and that it must always be attributed to the operation. Should a patient die of pneumonia either in the one stage or the two stage procedure, of emboli or of any other complication other than one directly related to the operation, it must, I believe, in all fairness and in order that one may correctly evaluate procedures, be attributed to the operative method.

In view of the fact that I have been able to deal satisfactorily with 118 of these patients, with but 1 fatality, it would be difficult for anyone to convince me of the wisdom, for me at least, of employing the one stage procedure.

As one compares the results and the thoroughness with which the operation can be done with either the one stage or the two stage procedure, there is little to be said in favor of either as relates to this aspect, One can dissect the sac and amputate the neck of the sac as completely in one as in the other. As far as reenforcing the muscular defect over the mucosa goes, in the one stage operation the muscles over the diverticulum are so flimsy that even though they can be sutured in the one stage operation I doubt that they add much in the way of support over the ligated neck.

With the exception of the method employed in 5 cases, in which the sac was completely dissected and suspended (fig. 9) and in which, owing to the condition of the patient, the second stage was not performed, all of the operations reported on here have been done in two stages. One patient died of pneumonia one month after discharge from the hospital. The remaining 4 patients, on whom the second stage of the operation was not performed, are in satisfactory condition at the end of six years, seven months and six months (2 patients) respectively.

I have repeatedly described the technic which I have employed in cases of esophageal diverticulum. I have employed a long longitudinal incision in front of the sternocleidomastoid muscle. This has been used in preference to a transverse goiter incision as advocated by others, because it gives wide exposure of the area to be dissected and because it permits high implantation of the sac and favors accurate and anatomic demonstration of all the structures to be dealt with. Any one who has operated on many of these pharyngoesophageal diverticula will be conscious of the fact that the success of this operation is related to the thoroughness with which the neck of the sac is completely freed of all of its covering muscle fibers. Unless the acute angulation of the neck of the sac is so completely freed that as the sac is carried upward this acute angle is converted into an obtuse one, the dissection is not complete. Unless all muscle fibers have been so removed that the pale white submucosa of the neck of the sac presents itself plainly, dissection has

not been complete. Unless the slinglike muscle fibers beneath the lowest angle of the neck of the sac have been slit there will constantly be a tendency toward recurrence of the sac at this point. It is, therefore, evident that no incision which in any way limits exposure is desirable in this operative procedure, fraught as it so often is with possibilities of serious technical complications, such as perforation of the sac, incomplete dissection of its neck, hemorrhage from the inferior thyroid artery, injury to the recurrent laryngeal nerve, tears in the internal jugular vein and injury to the superior cervical sympathetic ganglion, with the production of Horner's syndrome.

For the foregoing reasons it has always been the conviction of this clinic that the safety which results from adequate exposure and anatomic dissection in a field in which bleeding can be accurately controlled far outweighs the question of the appearance of the scar.

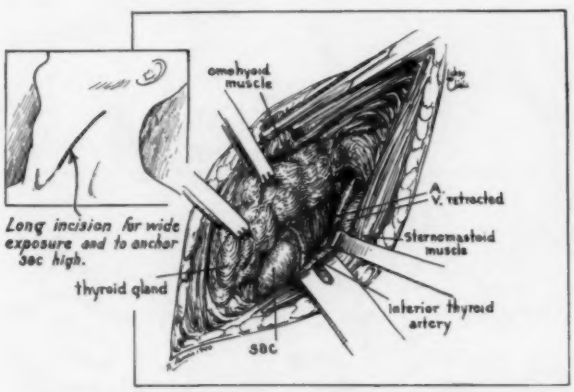


Fig. 7.—This and the following illustrations (8 and 9) demonstrate diagrammatically the technical procedures which have been employed in this clinic and some of which I have devised in the surgical treatment of pharyngoesophageal pulsion diverticula. In the insert may be seen the longitudinal incision in front of the sternocleidomastoid muscle. In the main illustration, the incision has been deepened so that the omohyoid muscle has already been severed, the internal jugular vein and the common carotid artery retracted, the thyroid gland mobilized toward the median line and the inferior thyroid artery with the sac of the diverticulum beneath it exposed. This illustrates also how necessary it is to divide the inferior thyroid artery in order that the sac beneath it may be dissected and mobilized.

The operative technic is well shown in the technical illustrations (figs. 7, 8 and 9). The longitudinal incision in front of the sternocleidomastoid muscle is carried through the skin down to the sternocleidomastoid muscle. This muscle is dissected back until the omohyoid muscle is well demonstrated. The omohyoid muscle is then severed at its upper attachment and at the point where it disappears beneath the sternocleidomastoid muscle; it is amputated at this point, and its stumps are

tied. With the omohyoid muscle out of the way, the thyroid gland is separated from the internal jugular vein and the common carotid artery, and the thyroid gland is picked up with blunt retractors and pulled toward the middle line. The inferior thyroid artery is then demonstrated deep in the neck, cut between forceps and ligated. Good-sized diverticulum sacs descend into the mediastinum beneath the inferior thyroid artery, so that it is necessary that that structure be severed in order that the delivery of such deep sacs may be safely accomplished. After the inferior thyroid artery has been tied and the lobe of the thyroid elevated, if the patient is asked to swallow the sac of the diverticulum will immediately be seen to ascend and descend. In most cases the sac will be closely bound to the longitudinal esophagus by the enveloping fibers of the cricopharyngei muscles. These are separated at the lowest

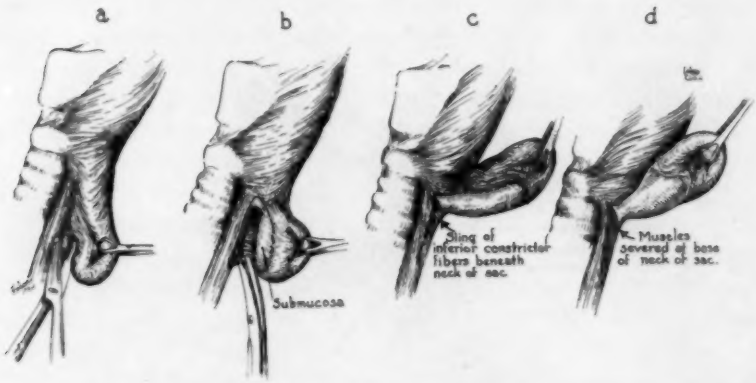


Fig. 8.—In illustration *a* may be seen diagrammatically the plan of separating the dependent sac of the diverticulum from the longitudinal esophagus, to which it is adherent and over which run the enveloping fibers of the cricopharyngei muscles. Note the sac grasped with the Babcock blunt forceps so that traction can be made on it to facilitate the separation. In illustration *b* the anterior fibers enveloping the wall of the sac have been separated, and the posterior fibers are now being cut. Note the appearance of the submucosa as the fibers running over the sac and enveloping it with the longitudinal esophagus are severed. Note particularly (illustration *c*) that the sac has been completely dissected up to its neck and that the sling fibers of the inferior constrictor muscle immediately beneath the neck of the sac have as yet not been severed. It is failure to sever these sling fibers that is so apt to bring about production of a shelf by means of which food is caught and recurrence of the herniation encouraged. In addition to this, failure to cut these sling fibers makes it impossible to mobilize the sac upward so completely that what was an acute angle at the lower aspect of the neck can be adequately converted into an obtuse angle. In illustration *d* may be seen the completely freed submucosa and the incision of the sling fibers of the inferior constrictor muscle immediately beneath the neck of the sac. This diagrammatic illustration demonstrates the completely mobilized sac with its submucosa and all of the muscle fibers about the neck of the sac freed and cut. It is now ready to be implanted high in the neck.

angle of the sac, and the dome of the sac is grasped with blunt Babcock forceps. The neck of the sac is then completely dissected.

As already has been stated, great care must be exercised in working out the line of cleavage between the inner lateral wall of the diverticulum sac and the outer longitudinal wall of the esophagus. Once this is established the dissection immediately becomes easier. As the sac is gradually dissected upward and freed it will become evident that, while the attachment of the lateral wall of the sac to the longitudinal esophagus in front has been freed, it is still bound down by the overlying muscle fibers in the back and behind the esophagus on the opposite wall. It is

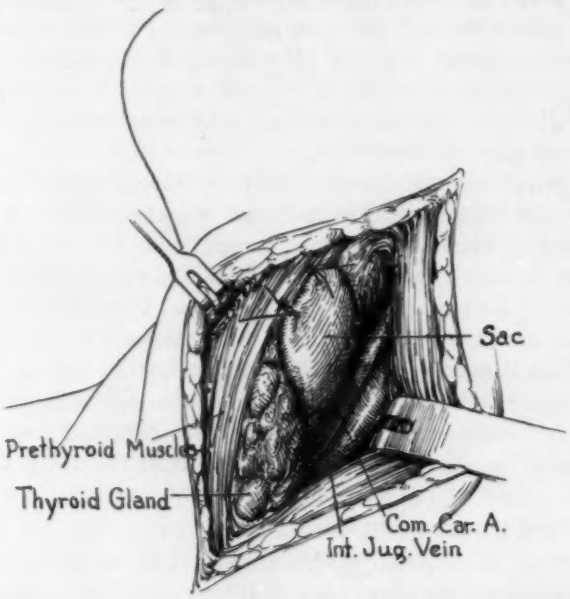


Fig. 9.—Plan by which the completely freed sac, as shown in illustration 8 *d*, is attached by black silk stitches through adventitious tissue over the wall of the sac to the prethyroid muscles in the neck at such a height that the dome of the sac is well above the level of the neck, thus preventing the entrance of food. After this, a gauze drain is inserted into the mediastinum, where it remains for four days. At the end of eight days, when the mediastinum and fascial planes are walled off, the wound is reopened; the dome of the sac is cut off; the mucosa is trimmed from within the sac, and the canal of submucosa without its mucous membrane (shown in this illustration) is preserved and packed with gauze. The gauze is removed on the fourth day, when the wound collapses and the submucosal canal usually closes with no drainage.

at this point that wide exposure is so necessary, since unless the muscles covering the sac which run behind the esophagus to the opposite side are freed it will not be possible to deliver the sac completely, so that it hangs entirely free by its neck.

With the sac hanging entirely by its neck it becomes extremely important to dissect with meticulous care all of the muscle fibers about the neck of the sac and particularly those fibers acting as a sling at the most inferior angle of the neck of the sac (fig. 8*c*). With the sac so completely dissected that it hangs freely by its neck with all overlying muscle fibers detached, with the slinglike muscle fibers under the lower angle of the neck of the sac slit and with the white submucosa of the sac neck clearly visible (fig. 8*d*), the dissection is complete. The sac is then carried upward over the upper pole of the thyroid and pinned by two black silk stitches to the uppermost fibers of the sternohyoid muscle. Care is to be taken in inserting these two black silk stitches that the needle does not pass through the wall of the sac but only through the adventitious tissue about the sac. If the needle is passed through the wall of the sac the stitches will cut out as a result of traction on the sac during swallowing, and there will be contamination of the wound and serious secondary infection in it.

When my associates and I first began to suspend these small sacs temporarily to the edge of the sternohyoid muscle within the wound merely by inserting black silk stitches through the adventitious tissue about the dome of the sac, we were fearful lest this rather delicate tissue would not keep the sac suspended in this position. There has never been any difficulty about this, in spite of the fact that with relatively small sacs there has undoubtedly been some traction on the sac. If the sac remains for two or three days fixed by the black silk stitches in this upward position it will become so adherent as a result of the exudate about it that even though moderate retraction from the stitches occurs the sac will be found at the second stage of the operation still in its proper location and pointing upward.

It is desirable to employ two black silk stitches to fix the dome of the sac to the sternohyoid muscles, leaving the ends about ½ inch (1.2 cm.) long. By this plan, which I suggested a few years ago, it is possible to find the sac quickly when the wound is reopened at the end of eight to ten days. Prior to the employment of this plan, owing to the fact that the granulating wound frequently oozed considerably, it was often not easy promptly to locate the sac within the wound.

After implantation of the sac, the gauze end of a good-sized cigaret drain is inserted into the mediastinum to produce walling-off granulations and is left in place for four to five days. At the end of this time it is removed, and at the end of eight to ten days the second stage of the operation is undertaken, without danger of cellulitis or mediastinitis from contamination of the contents of the sac.

The patients are permitted to be out of bed on the afternoon of the first operation. They are permitted to swallow food and fluids

immediately, and because the tip of the sac is implanted higher than the neck (fig. 9) food at once passes readily by the diverticulum opening into the true esophagus and so on down into the stomach. One can feed the patients on the table as soon as the first stage of the operation is completed and it can be demonstrated that any obstructive symptoms formerly present have completely disappeared.

At the end of eight to ten days, with the patient under intratracheal cyclopropane anesthesia (regional anesthesia or field block is not satisfactory because of the secondary reaction within the wound), the wound is reopened. The finger is inserted along the sinus still present at the lowest point of the wound, where the drain was removed. The edges of the skin are gradually pulled apart, and with a piece of gauze the sternocleidomastoid muscle, the internal jugular vein and the carotid artery are gently wiped outward throughout their entire course in the wound. With the wound widely opened, the black silk stitches demonstrating the tip of the sac are at once evident. The sac itself is then located; it is grasped with Allis forceps; the black silk stitches are cut out, and the sac is wiped out of the bed which it has molded for itself in the tissues until it is entirely free. The tip of the sac is then cut off, and the two layers, the mucosa and the submucosa, making up the wall of the sac immediately become plainly evident.

At the first stage operation the wall of the diverticulum sac will be almost as thin as a sheet of delicate writing paper, but at the second stage the submucosa will be so edematous that it will be of the thickness of a heavy sheet of blotting paper, and the submucosa will be found readily separable from the mucosa.

The submucosa may then be grasped with tacking forceps, and the mucosa can, with blunt scissors, be easily and completely separated from the canal of submucosa until it is entirely freed to the neck of the diverticulum. The mucosa is then cut off at the neck of the diverticulum; a small piece of gauze is inserted in the submucosal canal remaining. The drain is then brought out through the wound; buried stitches of plain catgut approximate the platysma and the subcutaneous fat, and the wound is closed with clips (fig. 10). The advantage of retaining the submucosal canal freed of its mucosa is that the canal, which still points upward, collapses after removal of the drain from it, and this prevents postoperative drainage of food through the wound and establishment of a sinus.

The drain within the canal of submucosa is removed at the end of four to five days, and, provided it has been possible to retain the submucosa complete within the canal, there is usually healing without leakage.

Some of the operative complications which can occur and some which have occurred in the experience of this clinic require mention. One must be careful in dissections of the neck of the diverticulum sac, since it is to be recalled that the lowest fibers of the inferior constrictor muscles are attached to the horn of the thyroid cartilage and that this is the level at which the neck of the diverticulum sac is located. One must remember that it is beneath these lowest fibers of the constrictor muscles that the recurrent laryngeal nerve enters the larynx to become intralaryngeal. Care must be exercised, therefore, in dissections of the neck of the sac

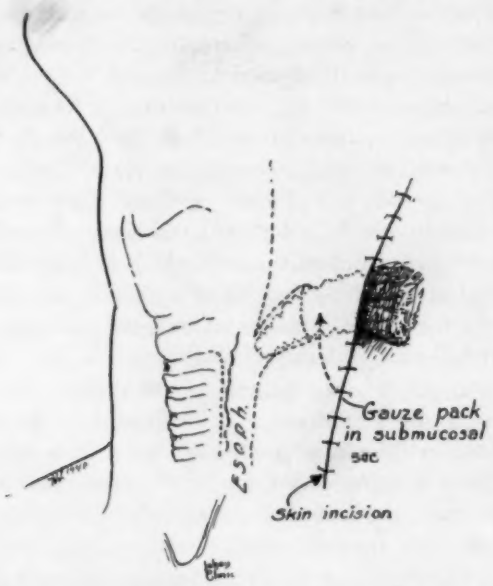


Fig. 10.—Diagrammatic illustration of the second stage of the operation, showing the gauze pack emerging into the upper part of the wound and placed through the submucosal canal, from which all mucosa has been removed. Note that the remaining submucosal canal points upward. When the gauze pack is removed, on the fourth day after operation, the canal will so collapse in its upward position that there will not be leakage of esophageal contents through it.

lest injury to the recurrent laryngeal nerve result. One must remember also that, as the common carotid artery and internal jugular vein are pulled back at the upper angle of the wound, not infrequently one comes in contact with the superior cervical sympathetic ganglion, injury to which will, of course, result in drooping of the lids and Horner's syndrome.

One must be careful with large sacs implanted in the wound with the tip of the sac above the level of the skin not to pull the sac too far out into the wound, lest the longitudinal esophagus be displayed

laterally and so acutely angulated that food cannot satisfactorily pass down its course. In my early experience this occurred to me, and on two occasions it required reopening of the wound and restoration of the dislocated esophagus to its midline position before adequate swallowing could be accomplished.

With large diverticula one must be careful to watch the sac implanted above the level of the skin within the dressing, because with the swallowing of food and air large diverticulum sacs not infrequently become so enormously distended and tense with air that gangrene can result. Should distention of the sac with air occur, a rubber catheter should, by means of a purse string suture, immediately be inserted into the sac through its tip to overcome this distention.

In no case in this entire series of 118 cases of pharyngoesophageal diverticulum has the sac, the neck of the sac or any level of the esophagus been perforated. I cannot, therefore, speak from experience as to what should be done should a perforation of the sac or of its neck occur during its dissection. I feel very strongly, however, that should it occur it should be carefully sutured with silk, with inversion of the point of perforation and accurate closure with lightly tied silk mattress sutures. I feel very strongly that, should leakage occur, there will always be the danger of contamination of the fascial planes and the mediastinum. In the event of this occurrence, large gauze packs should be inserted into the mediastinum and into the fascial planes behind the esophagus as a protection against the possible spread of the resulting infection downward toward the mediastinum, and, in addition, a feeding tube should be inserted through the nose into the stomach. One should not fail to recall that the esophagus with its diverticulum is in front of the prevertebral fascia and behind the pretracheal fascia and that this fascial plane leads directly into the superior mediastinum.

One cannot have operated on this number of esophageal diverticula without having dealt with almost all of the complications that can arise in connection with them. One of the most distressing is a persisting sinus through which food is discharged for several weeks after operation. Such a sinus is, in my opinion, most often the result of incomplete and inadequate dissection of the sac. It is most often the result particularly of inadequate dissections of the neck of the sac. If the angle shown in figure 8 *b* is not completely freed so that it is converted into the angle shown in figure 8 *d*, there will constantly be a ridge or shelf of mucous membrane which will catch food, discharging it outward along the sinus and thus keeping the sinus tract open. I can say from experience, however, that although I am sure that some of the sac necks in the operations mentioned were not dissected as completely in my early experience as I should today like to have them, none of the sinuses ultimately failed to close.

Recurrence took place in 3 of these cases, and reoperation was required. I am sure that the recurrences were the result of incomplete dissection of the neck of the sac before I had had the necessary experience with which to accomplish these painstaking dissections.

There has often been considerable debate as to whether patients with esophageal diverticula require postoperative dilation. A number of patients from considerable distances, operated on at this clinic, have not been able to obtain postoperative dilation and have had quite as satisfactory results as those on whom dilations have been done. This group, however, represents but a small proportion of the entire group, and so one cannot say with fairness that the results in all cases without postoperative dilation are as good as those in cases in which postoperative dilation is done. It has always seemed to me that one cannot carry out these operative procedures at the pharyngoesophageal junction either in one or in two stages without producing considerable scarring. Whether one does this operation in one stage or in two stages there must still be present a certain degree of muscular weakness at this level and perhaps some incoordination of the cricopharyngeal muscles, and for this reason it has always seemed wise to me when possible to do dilations after operation.

Postoperative dilation is done in this clinic by the laryngologists; a Plummer bag is used, and wide dilatation is carried out.

In reviewing my experience with pharyngoesophageal diverticula, my conclusions are: 1. At the very early stage, operation is not indicated. 2. It is desirable that these patients be submitted to operation once the sac is well formed, with a definite sac and a definite neck. It is undesirable to permit these sacs to reach such a size that they enter the mediastinum and produce obstructive symptoms. 3. The two stage operation in my hands satisfactorily fulfils the requirements of adequate removal of the sac with safety. 4. The most important technical features of the operation are that the neck of the sac be completely freed of all its muscular coverings until the pale submucosa is entirely visible about the neck of the sac; that the tip of the sac be so implanted upward that what was at first an acute angle at the inferior aspect of the neck of the sac becomes an obtuse angle so that food cannot be caught in it, and that the greatest care be exercised lest the sac or its neck be perforated during the dissection.

TRACTION DIVERTICULA

Figure 2 shows typical types of traction diverticula. These diverticula originate from inflammatory processes in adjacent bronchial lymph nodes. These inflammatory reactions become attached to the esophagus and, as cicatrization occurs, result in traction bands which pull the esophagus out in the lateral direction.

The symptoms associated with this type of diverticulum (traction) are rarely urgent. They consist largely of partial degrees of obstruction or interference with the progress of food and are as a rule promptly relieved by dilation.

Owing to the fact that most traction diverticula are pulled in either a lateral or an upward direction, they tend to empty themselves. Spontaneous emptying of these sacs is further favored by the fact that muscular contraction occurs within the walls of the sac. Since the walls of the sac are made up of all the coats of the esophagus, including the muscular coat, muscular expulsion of the contents of the sac is possible.

Operative treatment is not indicated for diverticula of this type and, if it were, would be extremely difficult to accomplish, since they are difficult to approach, located as they so often are at the bronchial hilus and not possessing necks. It must be recalled in surgical intervention on the esophagus that leaks are particularly apt to occur in esophageal suture; that the structure of the esophagus is not the best from the point of view of holding sutures, and that the blood supply of the organ is by no means profuse.

PULSION DIVERTICULA (SUPRADIAPHRAGMATIC)

The final type of diverticulum to be discussed is that shown in figure 3, the supradiaphragmatic pulsion diverticulum. These diverticula fortunately are extremely rare. In the fairly good-sized experience of this clinic with esophageal diverticula, only 4 have occurred.

These diverticula have well developed sacs with narrow necks, and their lateral walls tend to become adherent to the longitudinal esophagus, as do the lateral walls of the pharyngoesophageal pulsion diverticula.

The symptoms associated with the 4 cases of this type which have been observed in this clinic were related to decomposition of food which remained within such a large sac over a long period and regurgitation of such food during the night, interfering with sleep.

In a previous discussion of this type of diverticula, I described a conservative method of treatment which proved satisfactory. With the chest open and the lower lobe of the lung held out of the way, this type of diverticulum may be readily dissected so that it hangs freely by its neck. Owing to the fact, already stated, that the esophagus holds stitches badly and that amputation of the sac at its neck could be accomplished only at the risk of soiling of the pleural cavity, it was considered unwise in this case to attempt excision of the sac, and there appeared no plan by which it could be safely submitted to a two stage procedure.

The plan devised for this case and reported in 1933 consisted of fixing the dome of the completely freed sac with silk stitches high in the pleural gutter beside the vertebral bodies so that it was implanted

upward as a cord parallel to the longitudinal esophagus. The same principle was utilized in this procedure which is employed in the first stage operation for pharyngoesophageal pulsion diverticulum. With this plan the sac can be converted into a stringlike structure fixed by black silk stitches not passed through all of the walls of the sac and caught to the parietal pleura. With the sac so implanted high in the pleura beside the esophagus, food, as can be demonstrated roentgenographically, passes readily by the neck of the sac and can be made to enter the sac only when the patient is placed in the Trendelenburg position. This is a useful procedure to employ in these cases, by means of which the sac can be made to remain free from accumulated food without the hazard of pleural contamination by its removal.

CONSERVATIVE TREATMENT OF ACHALASIA

ELMER B. FREEMAN, M.D.
BALTIMORE

Success of conservative treatment of achalasia depends mostly on complete dilation of the cardia and not on drug therapy or dietetic management. Those interested in the conservative treatment of achalasia are in complete agreement that some type of dilation of the cardia is needed to overcome the obstruction, but there is a very wide difference of opinion as to the procedure by which this is to be accomplished. Each of the following methods has its advocates:

(1) Dilation with mercury-filled bougies; (2) dilation with bougies passed through the esophagoscope; (3) dilation with the combined mercury bougie and pneumatic dilator; (4) dilation with a pneumatic or hydrostatic dilator, and (5) dilation under fluoroscopic control.

It is not claimed by the advocates of any of the procedures that the dilated esophagus will return to normal after thorough dilation of the cardia, even though the obstructive symptoms have been completely relieved. As a matter of fact, it has been definitely proved that the dilated esophagus never regains its normal tone. But it is maintained by advocates of each of the procedures that the obstruction can be sufficiently overcome in most cases to permit the contents of the esophagus to pass freely through the cardia.

DILATION WITH MERCURY-FILLED BOUGIES

The mercury-filled bougies as designed by Hurst consist of a number of rubber tubes of different sizes, each filled with the same amount of mercury, i. e., 1 lb. 5 ounces (609 Gm.). The bougies range in size from approximately ½ inch (1.2 cm.) in diameter to ¾ inch (1.9 cm.) or even more. After the bougie has been introduced into the esophagus, the weight of the mercury carries it through the cardia. Sometimes the bougie is passed under fluoroscopic control, when it is considered desirable to determine its exact relation to the cardia. In many cases it is necessary to introduce the bougie daily in order to control the obstruction and keep the cardia sufficiently dilated to permit the free passage of esophageal contents. In some cases of severe involvement it is necessary to introduce the bougie before every meal. This method has one very decided advantage, in that the bougie is easy to introduce and the patient may be readily trained to introduce it himself. After the treatment has been established, the size of the bougie and the frequency of introduction in many cases may be left to the discretion of the patient.

DILATION WITH BOUGIES PASSED THROUGH THE ESOPHAGOSCOPE

Dilation of the cardia with bougies passed through the esophagoscope is not, in my opinion, a satisfactory procedure. First, it necessitates the introduction of the esophagoscope whenever it is deemed necessary to give a treatment. This in itself is an ordeal. With the esophagoscope in position, the introduction of a large bougie through the instrument so obstructs vision that one cannot keep the bougie under careful observation while it is being introduced. The main objection is, however, that it is impossible to pass a bougie of sufficient size through an esophagoscope to dilate the cardia completely, which is necessary if one is to overcome the obstruction and thus relieve the symptoms.

DILATION WITH THE COMBINED MERCURY BOUGIE AND PNEUMATIC DILATOR

Browne and McHardy have recently devised a mercury bougie with a pneumatic dilator which embraces many of the good qualities of the Hurst, Plummer and Smithies instruments. They have frankly stated: "This dilator is not presented with the idea that it is applicable in all instances, any more than other instruments may meet all emergencies, but rather that it fills a need most frequently encountered."

DILATION WITH A PNEUMATIC OR HYDROSTATIC DILATOR

Dilation of the cardia with either a pneumatic or a hydrostatic dilator is, I believe, the most satisfactory method available at present to overcome the resistance at the lower end of the esophagus that is met with in cases of achalasia. In my opinion it does not make any material difference which method is used; it is a matter of personal preference. The dilating bag should be of sufficient caliber to dilate the cardia completely, and the dilator should be so designed that it may be easily introduced and will maintain its position in the cardia while the dilating bag is being distended. In my opinion, the Plummer modification of the Russell dilator, with either air or water used to distend the dilating bag, fulfils these requirements more satisfactorily than does any other instrument so far designed. Certain precautions, however, should be observed. First, one should pass a no. 60 French scale bougie over a previously swallowed, properly anchored thread as a guide. If a no. 60 bougie cannot be passed without difficulty, the probability is that the obstructive symptoms are due not to dysfunction of the lower end of the esophagus but to an organic lesion of the esophagus or an organic lesion of the cardiac end of the stomach encroaching on the cardia. It is therefore most important to determine whether a large bougie will pass easily through the cardia. If one cannot pass a large bougie, no attempt

should be made to introduce the Plummer dilator. It is also very important to pass the dilator over a previously swallowed, properly anchored size D silk thread. The size of the thread is likewise very important. Experience extending over many years has definitely proved that size D thread is the most satisfactory to use. For the thread to become properly anchored it is necessary to swallow 6 yards (5.4 meters) of it very slowly the night before the treatment is to be given. This permits the thread to work its way through the stomach and become anchored in the small intestine. The end protruding from the mouth should be fastened to the clothing, which makes it available when desired. After the treatment has been given, the thread is cut and permitted to pass on through the gastrointestinal tract. Nothing is given by mouth except water or weak tea from the time the patient starts to swallow the thread until the treatment is given. If these simple precautions are followed, one should not have any difficulty in introducing the instrument. It is not at all necessary to introduce the instrument under fluoroscopic control, as by the sense of touch one can easily tell when the instrument is in proper position. If it tends to slip downward into the stomach or upward into the esophagus when the dilating bag is being distended, it indicates a faulty position of the instrument, which can be readily corrected by releasing the pressure and changing the position as indicated. The number of dilations necessary to relieve the obstruction cannot be determined until after treatment has been begun. In many cases almost complete relief is obtained after one satisfactory dilation; in others several treatments may be required, and in an occasional one a treatment may be needed every month or two, or even at longer intervals, to keep the patient comparatively free of symptoms. The amount of pressure necessary to obtain relief of symptoms varies from the equivalent of 10 to 18 feet (3 to 5.4 meters) of water pressure or even more. The end results with the Plummer treatment are satisfactory in at least 75 to 80 per cent of the cases. In most of the remaining 20 to 25 per cent the patient is materially benefited.

DILATION UNDER FLUOROSCOPIC CONTROL

With few exceptions, there is no advantage in dilating the cardia under fluoroscopic control. However, if one is dilating the cardia with mercury-filled bougies, it may be helpful in determining the exact relation of the bougie to the cardia. If the dilator is introduced over a previously swallowed thread, there is no necessity for fluoroscopic control, as by the sense of touch alone one is able to determine when the dilator is in proper position. Dilation under fluoroscopic control necessitates working in a dark room, which, in my opinion, is a distinct disadvantage, in that one is not able to observe the patient while the dilation

is being done. I have always relied very much on my observation of the patient while the dilating bag is being distended. By observation at that time one is able, in a measure at least, to determine how much pain the dilation is causing. The degree of pain produced is helpful in determining the amount of pressure to be used. If the dilation causes much pain, one should be very cautious and increase the pressure slowly at subsequent treatments.

LOCAL TREATMENT

No local treatment of the associated chronic esophagitis, which is present in practically all cases of achalasia of the cardia to a greater or less degree, is required. Just as soon as the cardia has been sufficiently dilated to permit the esophageal contents to pass readily into the stomach, the esophagitis promptly disappears. Of course, early in the treatment one may be justified in resorting to lavage of the esophagus if there is marked dilatation with retention, but even this is not necessary after one or two complete dilatations of the cardia.

DRUG THERAPY

The administration of drugs is of questionable value. While antispasmodic sedative drugs seem to help to a certain degree, I think that their beneficial results are due to their general sedative effect and not to any local effect on the esophagus. If marked malnutrition or anemia is present, appropriate drug treatment is, of course, indicated.

DIET

Diet plays a relatively unimportant part in the treatment of achalasia of the cardia. It is a well established fact that early in the disease an attack of dysphagia is just as likely to occur while the patient is drinking liquids as while he is partaking of solid food. In the case of advanced involvement with marked dilatation of the esophagus and retention of food, it is well to restrict the diet to liquid foods mostly until a satisfactory dilation has been accomplished. Just as soon as there is complete dilatation of the cardia, which frequently follows the first treatment, esophageal contents will pass through the cardia freely, and no further restriction in diet is necessary. After the first satisfactory treatment, all dietary restrictions should be discontinued and the patient directed to follow a regular diet of his own selection. It is surprising to see how frequently he returns in a few days with the statement that he has had very little difficulty, the obstructive symptoms having almost entirely disappeared.

SUMMARY

For many years the Plummer modification of the Russell cardiospasm dilator has been used, with satisfactory results. Air instead of water

may be used to distend the dilating bag, as was advised by Plummer. I believe that the success of the treatment depends entirely on complete dilation of the cardia. I do not believe that this can be accomplished by the passage of mercury-filled bougies or by the passage of bougies through an esophagoscope, owing to the fact that neither of these procedures completely dilates the lower end of the esophagus. I also believe that complete dilation can be accomplished only by an instrument with a dilating bag sufficiently large, yet not too large, to dilate the cardia completely. Of all the different types of instruments that have been devised, I believe the one best adapted for the purpose is the Plummer cardiospasm dilator. I further believe that all patients with achalasia of the cardia should be given the benefit of thorough dilation before being considered from an operative standpoint. If patients are given the benefit of thorough dilation, few will require surgical intervention.

SURGICAL CONSIDERATIONS OF ACHALASIA

REVIEW OF THE LITERATURE AND REPORT OF THREE CASES

ALTON OCHSNER, M.D.
AND
MICHAEL DEBAKEY, M.D.
NEW ORLEANS

A variety of terms have been applied to the clinical syndrome characterized by dilatation and hypertrophy of the esophagus associated with nonorganic obstruction of the cardia. These include cardiospasm, achalasia, phrenospasm, idiopathic dilatation of the esophagus, esophagectasia, hiatal esophagismus, megaesophagus, simple ectasia of the esophagus, preventriculosis, dilatio ingluviformis oesophagi, dilatio fusiformis and dolichoesophagus. The multifarious designations clearly reveal the controversial causation and the bewildering pathogenesis of the condition. This is further reflected by the various types of therapeutic procedures which have been employed. It is considered inopportune to attempt here a detailed discussion of the different theories and concepts of the malady, as these have been adequately reviewed in previous publications.[1] However, it may be desirable to present briefly some of the factors which have been considered of pathogenic significance, in order to permit a better comprehension of the varied surgical therapeutic procedures that have been employed and advocated.

Purton (1821)[2] is usually credited with the first description of this condition, although attention has recently been directed to a report of

From the Department of Surgery, School of Medicine, Tulane University.

1. (*a*) Thieding, F.: Ueber Cardiospasmus, Atonie und "idiopathische" Dilatation der Speiseröhre, Beitr. z. klin. Chir. **121**:237, 1921. (*b*) Bull, P. N.: So-Called Idiopathic Dilatation of the Esophagus, Acta chir. Scandinav. **58**:581, 1925; (*c*) So-Called Idiopathic Dilatation of the Esophagus, Ann. Surg. **81**:59 and 470, 1925. (*d*) von Hacker, V., and Lotheissen, G.: Chirurgie der Speiseröhre, in von Bruns, P.: Neue deutsche Chirurgie, Stuttgart, Ferdinand Enke, 1926, vol. 34. (*e*) Cardenal, L.: La dilatación del esófago sin estenosis orgánica (el llamado cardioespasmo), Cong. Soc. internat. de chir., Rap. **1**:701, 1932. (*f*) Freeman, E. B.: Chronic Cardiospasm: Report of Fatal Case with Pathological Findings, South. M. J. **26**:71, 1933. (*g*) Sturtevant, M.: Cardiospasm with a Review of the Literature, Arch. Int. Med. **51**:714 (May) 1933. (*h*) Bird, C. E.: Recent Advances in Surgery of the Esophagus, Surgery **6**:617, 1939. (*i*) Flynn, R.: Achalasia of the Oesophagus, Australian & New Zealand J. Surg. **8**:244, 1939. (*j*) Sodeman, W. A.: Cardiospasm or Achalasia of the Esophagus, Am. J. M. Sc. **199**:132, 1940.

2. Purton, T.: An Extraordinary Case of Dilatation of the Esophagus, Forming a Sac, Extending from Two Inches Below the Pharynx to the Cardiac Orifice of the Stomach, London M. & Physiol. J. **46**:540, 1821.

a typical case by Willis [3] approximately a century and a half earlier. This astute observer not only foreshadowed one of the most popular forms of present day therapy but divined its possible causation in the statement that "the mouth of the stomach being always closed, whether by a tumor or palsie, nothing could be admitted into the ventricle unless it were violently opened." This concept is, in essence, similar to that of Einhorn,[4] who in 1888 concluded that the cardia probably fails to open reflexly during the act of deglutition, or to that of Hurst, who subsequently coined the term "achalasia" and popularized the idea of "absence of relaxation of the cardiac sphincter." [5] Rolleston [6] had expressed the same opinion. Rake [7] and others [8] demonstrated degenerative changes in the ganglion cells of Auerbach's plexus at the lower end of the esophagus in cases of achalasia. On the basis of these alterations, Hurst [9] expressed the belief that the condition is the result of some organic change involving neuromuscular control of the sphincter. In fact, he stated the opinion that achalasia of the cardia is the "only well established example of localized disease of the autonomic nervous system." Of particular interest in this connection is the possibility of

3. Willis, T., cited by Hurst.[9]

4. Einhorn, M.: A Case of Dysphagia with Dilatation of the Esophagus, M. Rec. **34**:751, 1888.

5. (*a*) Hurst, A. F., in discussion on Weber: Spasmodic Stricture of Oesophagus, Proc. Roy. Soc. Med. (Clin. Sect.) **7**:150, 1913; (*b*) Case of Achalasia of the Cardia (So-Called Cardiospasm), ibid. **8**:22, 1914; (*c*) Reports of Royal Society of Medicine, Brit. M. J. **2**:1062, 1914; (*d*) Les spincters du canal alimentaire et leur signification clinique, Arch. d. mal. de l'app. digestif **15**:1, 1925; (*e*) Treatment of Achalasia of the Cardia, Lancet **1**:618, 1927. (*f*) Hurst, A. F., and Rake, G. W.: Achalasia of the Cardia (So-Called Cardiospasm), Quart. J. Med. **23**:491, 1930.

6. Rolleston, H. D.: Simple Dilatation of the Oesophagus, Tr. Path. Soc. London **47**:37, 1896.

7. Rake, G. W.: A Case of Annular Muscular Hypertrophy of the Oesophagus (Achalasia of the Cardia Without Oesophageal Dilatation), Guy's Hosp. Rep. **6**: 145, 1926; On the Pathology of Achalasia of the Cardia, ibid. **7**:141, 1927.

8. (*a*) Cameron, J. A. M.: Oesophagectasia in a Child, Arch. Dis. Childhood **2**:358, 1927. (*b*) Mosher, H. P., and McGregor, G. W.: A Study of the Lower End of the Esophagus, Ann. Otol., Rhin. & Laryng. **37**:12, 1928. (*c*) Hara, H. J.: Cardiospasm, California & West. Med. **30**:390, 1929. (*d*) Beattie, W. J. H. M.: Achalasia of the Cardia, St. Barth. Hosp. Rep. **64**:39, 1931. (*e*) Scrimger, F. A. C.: Idiopathic Dilatation of the Esophagus, Ann. Surg. **94**:801, 1931. (*f*) Etzel, E.: Neuropathologia do megaesôfago e megacolo. Estudo de 5 casos, Ann. Fac. de med. da Univ. de São Paulo **10**:383, 1934; (*g*) Os nervos pneumogástricos no megaesôfago. Estudo anatomopatológica de 8 casos, ibid. **10**:373, 1934. (*h*) Lendrum, F. C.: Anatomic Features of the Cardiac Orifice of the Stomach with Special Reference to Cardiospasm, Arch. Int. Med. **59**:474 (March) 1937. (*i*) Janes, R. M., in discussion on Gray and Skinner. [5711]

9. Hurst, A. F.: Some Disorders of the Esophagus, J. A. M. A. **102**:582 (Feb. 24) 1934.

a chronic vitamin B_1 deficiency as the cause of the degeneration of Auerbach's plexus.[10]

In other attempts to explain the pathogenesis on the basis of a nervous mechanism the vagus nerve has been considered. Numerous investigators [11] have suggested a reflex irritability of the vagus nerve, which may be primary [12] or secondary, due to a floating kidney,[13] poorly masticated food [14] or trauma and infection.[15] Whereas some observers [16] have described degenerative lesions in the vagus nerve, others [17] have found no pathologic changes. As early as 1839 it was shown

10. Etzel, E.: Megaesôfago-megacolon y sus asociaciones morbidas, Rev. de cir. de Buenos Aires **14**:631, 1935; A avitaminose como agente etiologico do megaesôfago e do megacolo, Ann. Fac. de med. da Univ. de São Paulo **11**:59, 1935; Megaesophagus and Its Neuropathology: A Clinical and Anatomo-Pathological Research, Guy's Hosp. Rep. **87**:158, 1937; footnotes 8 *f* and *g*.

11. von Openchowsky, T.: Ueber die gesammten Innervation des Magens, Deutsche med. Wchnschr. **15**:717, 1889; **23**:48, 1897. Tyson, J.; Martin, E., and Evans, J. S., Jr.: Diffuse Dilatation of the Esophagus Due to Cardiospasm, New York M. J. **80**:731, 1904. Smukler, M. E.: Cardiospasm with Dilatation of the Esophagus, ibid. **99**:772, 1904. Carlson, A. J.; Boyd, T. E., and Pearcy, J. F.: Studies on the Visceral Sensory Nervous System: XIV. The Reflex Control of the Cardia and Lower Esophagus in Mammals, Arch. Int. Med. **30**:409 (Oct.) 1922.

12. Held, I. W., and Gross, M. H.: Cardiospasm, J. A. M. A. **66**:233 (Jan. 22) 1916.

13. Lockwood, C. B.: Case of (So-Called) Idiopathic Dilatation of the Oesophagus, Brit. M. J. **1**:1367, 1903.

14. Guisez, J.: De l'étiologie et pathogénie des spasmes primitifs et graves de l'œsophage, Bull. Acad. de méd., Paris **83**:147, 1920; Pathogénie et traitement des grandes dilatations de l'œsophage, Presse méd. **29**:661, 1921.

15. von Bergmann: Ein Fall von Dilatatio oesophagi idiopathica, Berl. klin. Wchnschr. **45**:330, 1908. Meyer, H.: Entstehung und Behandlung der Speiseröhrenerweiterungen und des Cardiospasmus, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **34**:484, 1922. Stephan, cited by Fischer, W., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 4, p. 101.

16. (*a*) Kraus, F.: Die Erkrankungen der Speiseröhre, in Nothnagel, H.: Specielle Pathologie und Therapie, Vienna, A. Hölder, 1902, vol. 16, pt. 1, sect. 2, no. 1, p. 129. (*b*) Heyrovsky, H.: Casuistik und Therapie der idiopathischen Dilatation der Speiseröhre, Oesophago-gastroanastomose, Arch. f. klin. Chir. **100**:703, 1912-1913. (*c*) Pollitzer, H.: Idiopathischen spindelförmigen Erweiterung des Oesophagus, München. med. Wchnschr. **60**:108, 1913. (*d*) Loeper, M., and Forestier, J.: Les lésions nerveuses du pneumogastrique et le cardiospasme récurrent dans le cancer de l'estomac, Arch. d. mal. d. l'app. digestif **11**:307, 1921.

17. (*a*) Rosenheim, T.: Beiträge zur Oesophagoskopie, Deutsche med. Wchnschr. **24**:53 and 75, 1899. (*b*) Schmidt, M. B.: Ueber idiopathische Oesophaguserweiterung, ibid. **31**:1522, 1905. (*c*) Ewald, C. A.: Idiopathische spindelförmige Erweiterung der Speiseröhre, ibid. **33**:1036, 1907. (*d*) Starck, H.: Zur Pathologie der Erweiterungen der Speiseröhre mit besonderer Berücksichtigung des Röntgenverfahrens, Verhandl. d. deutsch. Kong. f. inn. Med. **29**: 122, 1912. (*e*) Harbitz, F.: Idiopathic Dilatation of the Esophagus, Norsk mag. f. lægevidensk. **79**:841, 1918.

experimentally in rabbits that vagal section in the neck resulted in stasis of food in the esophagus.[18] Subsequent investigators[19] expressed the opinion that this was due to an increased tonus of the cardiac sphincter. Some investigators[20] have observed that in cats as well as in dogs bilateral vagal section or destruction produced obstruction and dilatation of the esophagus, whereas others[21] have not. In addition to the lack of uniformity of opinion concerning the vagal control of the esophagus, there are conflicting views concerning the influence of the sympathetic nervous system on the esophagus. Whereas some investigators[22] have obtained definite response to stimulation or section of the sympathetic nerve supply, others[23] have not. Knight,[22e] who reviewed and investigated this subject, has shown that apparently some

18. Reid, J.: An Experimental Investigation into the Functions of the Eighth Pair of Nerves, or the Glosso-Pharyngeal, Pneumogastric, and Spinal Accessory, Edinburgh M. & S. J. **51**:269, 1839.

19. Bernard: Paralysis de l'œsophage par la section des deux nerfs pneumogastriques, Compt. rend. Soc. de biol. **1**:14, 1850. Schiff, J. M.: Leçons sur la physiologie de la digestion, Florence, Hermann Loescher, 1867, p. 350. Kronecker, H., and Meltzer, S.: Der Schluckmechanismus. Seine Erregung und seine Hemmung, Arch. f. Anat. u. Physiol., 1883, supp., p. 328. Klee, P.: Der Einfluss der Vagusreizung auf den Abbau der Verdauungsbewegungen. Röntgenversuche an der Rückenmarkskatze, Arch. f. d. ges. Physiol. **145**:557, 1912. Ozorio de Almeida, M.: Sur la dilatation chronique de l'œsophage produite expérimentalement chez le chien par section partielle des pneumogastriques, Compt. rend. Soc. de biol. **102**: 407, 1929.

20. (*a*) Cannon, W. B.: Esophageal Peristalsis After Bilateral Vagotomy, Am. J. Physiol. **19**:436, 1907. (*b*) Carlson, A. J.; Boyd, T. E., and Pearcy, J. J.: Studies on Visceral Sensory Nervous Innervation of Cardia and Lower End of Esophagus in Mammals, ibid. **61**:14, 1922. (*c*) Kelly, U. B.: Nervous Affections of the Esophagus, J. Laryng. & Otol. **42**:221, 1927. (*d*) de Vasconcellos, E.: L'œsophagogramme du chien normal et du chien porteur d'un mégacœsophage expérimental, Compt. rend. Soc. de biol. **116**:1128, 1934. (*e*) Grondahl, J. W., and Haney, H. F.: Attempt to Produce Experimentally Cardiospasm in Dogs, Proc. Soc. Exper. Biol. & Med. **44**:126, 1940.

21. Krehl, L.: Ueber die Folgen der Vagusdurchschneidung, Arch. f. Anat. u. Physiol., 1892, supp., pp. 278-290. Sinnhuber: Beiträge zur Lehre vom musculären Cardiaverschluss, Ztschr. f. klin. Med. **50**:102, 1903. Zeller, W., and Burget, G. E.: A Study of the Cardia, Am. J. Digest. Dis. & Nutrition **4**:113, 1937.

22. (*a*) Carlson, A. J., and Luckhardt, A. B.: Visceral Sensory Nervous System; Vagus Control of Esophagus, Am. J. Physiol. **52**:299, 1921. (*b*) Kuré, K.: Experimentelles Studium über die Innervation des Oesophagus, Arch. f. d. ges. Physiol. **221**:367, 1929. (*c*) Knight, G. C.: The Relation of the Extrinsic Nerves to the Functional Activity of the Esophagus, Brit. J. Surg. **22**:155, 1934. Carlson and others.[20b]

23. (*a*) Page-May, W.: The Innervation of the Sphincters and Musculature of the Stomach, J. Physiol. **31**:260, 1904. (*b*) Jurica, E. J.: Motility of Denervated Mammalian Esophagus, Am. J. Physiol. **77**:371, 1926. (*c*) Rieder, W.: Der sogenannte Cardiospasmus, Deutsche Ztschr. f. Chir. **217**:334, 1929.

of this confusion is due to the type of experimental animal employed, as the type and amount of muscle vary. Cats were employed in his experiments, because the esophagus of the cat corresponds closely to that of the human being. He demonstrated in the interdiaphragmatic and intra-abdominal portions of the esophagus an intrinsic sphincter mechanism with relaxation on stimulation of the vagus nerves and contraction on stimulation of the sympathetic nervous system. Bilateral vagotomy resulted in the clinical manifestations of achalasia, which could be prevented by sympathectomy. These findings after vagotomy were confirmed in monkeys by Ferguson.[24]

Another concept completely antithetic to the theory of achalasia is reflected by the term cardiospasm, which was popularized by von Mikulicz,[25] in 1882, although Huss,[26] forty years previously, had propounded a similar theory. On the basis of this concept the dilatation of the esophagus is secondary to a true spasm of the cardia.[27] Accordingly it must be assumed that a cardiac sphincter exists. Whereas a number of observers [28] have maintained that such a sphincter is present, others [28a] have even more convincingly demonstrated its absence. It is indeed difficult to understand how such conflicting opinions can be expressed regarding an anatomic structure. But, as was pointed out by Sodeman,[1j] equally strange is the fact that only patients with achalasia should seek out one investigator and only those with cardiospasm another.

---

24. Ferguson, J. H.: Effects of Vagotomy on the Gastric Function of Monkeys, Surg., Gynec. & Obst. **62**:689, 1936.

25. von Mikulicz, J.: Ueber Gastroskopie und Oesophagoskopie, Mitth. d. Ver. d. Aerzte in Nied. Oesterreich **8**:41, 1882.

26. Huss, M.: Dilatatio oesophagi ingluviformis, Hygiea **4**:296, 1842.

27. Meltzer, S. G.: Ein Fall von Dysphagie, Berl. klin. Wchnschr. **25**:140, 1888.

28. Cannon, W. B.: The Mechanical Factors of Digestion, London, Edward Arnold & Company, 1911. Schreiber, cited by Sturtevant.[1g] Todd, T. W.: The Clinical Anatomy of the Gastro-Intestinal Tract, London, Longmans, Green & Company, 1915. Bennett, T. I.: The Stomach and Upper Alimentary Canal in Health and Disease, London, William Heinemann, 1925. Feldman, M., and Morrison, S.: An Experimental Study of the Lower End of the Esophagus: The Effect of the Diaphragm upon the Esophagus and Cardio-Esophageal Orifice in the Normal and Bilaterally Phrenicectomized Animal; Physiological Study, Am. J. Digest. Dis. & Nutrition **1**:471, 1934. Thieding.[1a] Hurst.[5e]

28a. Jackson, C.: Bronchoscopy and Esophagoscopy, ed. 2, Philadelphia, W. B. Saunders Company, 1927. Plenk, H.: Der Magen, in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1927, vol. 5, pt. 1. Kishi, S.: Ueber die Oesophagusmuskulatur, Arch. f. jap. Chir. **12**:851, 1935. Fleiner, W.: Neue Beiträge zur Pathologie der Speiseröhre, München. med. Wchnschr. **47**:529, 1900. Zaaijer, J. H.: Cardiospasm in the Aged, Ann. Surg. **77**:615, 1923. Mosher and McGregor.[8b] Lendrum.[8h]

Another widely held theory, originally postulated by Sauerbruch and von Hacker [29] and subsequently supported by others,[30] incriminates the diaphragm. Accordingly there is overactivity, a failure of relaxation or an incoordination of the diaphragmatic crura. Jackson [30c] suggested the term "phrenospasm," and others have supported this concept. Still other extraesophageal lesions have been suggested as having pathogenic significance. Handford,[31] in 1888, on the basis of a single observation, expressed the opinion that compression between a dilated aorta and the diaphragm may be the cause. Mosher,[32] who apparently conceives theories with great facility, has on various occasions suggested that the obstruction is produced by pressure on the esophagus as it passes through the hepatic tunnel or by the tips of the lungs by kinking and torsion of the redundant esophagus due to periesophageal or intramural fibrosis. Numerous other concepts have been propounded, such as "simple ectasia," [33] "congenital tendency," [34] trauma,[35] psychasthenia [36] and habitus.[37]

29. Sauerbruch, F., and von Hacker, R.: Zur Frage des Cardiaverschlusses der Speiseröhre, Deutsche med. Wchnschr. **32**:1263, 1906.

30. (*a*) Bassler, A.: Cardiospasm: What Is It? What It Seems to Be, New York State J. Med. **14**:9, 1914. (*b*) Hill, W., in Discussion on Dilatation of Esophagus Without Anatomical Stenosis, Proc. Roy. Soc. Med. (Sect. Laryng.) **12**:33, 1919. (*c*) Jackson, C.: The Diaphragmatic Pinchcock in So-Called "Cardiospasm," Laryngoscope **32**:136, 1922. (*d*) Caballero, R. V.: Étude expérimentale de la fermeture de l'extrémité inférieure de l'œsophage, Compt. rend. Soc. de biol. **88**:1060, 1923. (*e*) Joannides, M.: Influence of the Diaphragm on the Esophagus and on the Stomach, Arch. Int. Med. **44**:856 (Dec.) 1929. (*f*) Peroni, A.: Considerazioni cliniche e patogenetiche su alcune discinesie dell'esofago, Arch. ital. di otol. **49**:145, 1937.

31. Handford, H., cited by Hurst and Rake.[5f]

32. Mosher, H. P.: (*a*) Liver Tunnel and Cardiospasm, Laryngoscope **32**: 348, 1922; (*b*) Cardiospasm, Pennsylvania M. J. **26**:240, 1923; (*c*) The Lower End of the Esophagus at Birth and in the Adult, J. Laryng. & Otol. **45**:161, 1930; (*d*) Fibrosis of the Terminal Portion of the Esophagus: Cardiospasm, Proc. Internat. Assemb. Inter-State Post-Grad. M. A. North America **6**:95, 1931; (*e*) Involvement of the Esophagus in Acute and in Chronic Infection, Arch. Otolaryng. **18**:563 (Nov.) 1933.

33. Rosenheim.[17a] Mosher.[32a, b]

34. Zenker, F. A., in von Ziemssen, H.: Handbuch der speciellen Pathologie und Therapie, Leipzig, F. C. W. Vogel, 1876, vol. 7, pt. 1 (supp.). Mackenzie, M.: A Manual of Diseases of the Throat and Nose, London, J. & A. Churchill, 1884, vol. 1, p. 117. von Luschka, H.: Die spindelförmige Erweiterung der Speiseröhre, Virchows Arch. f. path. Anat. **42**:429 and 473, 1868. Sievers, R.: Zur Kenntnis der idiopathischen Oesophagus-Erweiterung, Ztschr. f. klin. Med. **49**:45, 1903. Fleiner, W.: Neue Beiträge zur Pathologie der Speiseröhre, München. med. Wchnschr. **47**:529, 1900.

35. Vinson, P. P.: External Trauma as a Cause of Lesions of the Esophagus, Am. J. Digest. Dis. & Nutrition **3**:457, 1936. Thieding.[1a]

(Footnotes continued on next page)

It becomes obvious from this brief review of the pathogenesis that there are considerable diversity of opinion and conflict of views regarding the development of this condition. There is little wonder, therefore, that there should be such great variation in the therapeutic measures advocated and employed. In general these procedures may be classified into the conservative and the radical. In this presentation no attempt will be made to discuss the former, although it should be realized that they should always be attempted first. It is generally agreed that the radical procedures should be instituted only after the conservative measures have failed.

The various types of radical procedures which have been advocated and employed may be classified as follows:

I. Operations directed at the dilated esophagus
   - (*a*) Excision of wall (Jaffé, Reisinger)
   - (*b*) Esophagoplication (Freeman, Reisinger, Meyer)
   - (*c*) Esophagostomia thoracica (Zaaijer)

II. Operations directed at the cardia
   - (*a*) Dilatation
     1. Retrograde
     2. Transgastric (von Mikulicz)
   - (*b*) Plastic surgical handling
     1. Cardiomyotomy (Heller, Röpke)
     2. Cardioplasty (Marwedel, Wendel)
   - (*c*) Excision
     1. Cardiectomy (Rumpel, Pribram)
   - (*d*) Deviation
     1. Esophagogastrostomy (Heyrovsky)

III. Operations directed at the diaphragm
   - (*a*) Phrenotomy
   - (*b*) Transposition

IV. Operations directed at the nerve supply
   - (*a*) Vagectomy (Meyer, and Sauerbruch)
   - (*b*) Sympathectomy

Because of the marked dilatation of the esophagus a procedure suggesting itself as corrective would be one which would decrease the size

36. Schindler, R.: Wesen und Behandlung der Cardiospasmus, München. med. Wchnschr. **73**:1612, 1926. Weiss, E.: Personality Study in Cardiospasm: The Meaning of the Disorder from the Standpoint of Behavior, Am. J. Digest. Dis. & Nutrition **3**:1, 1936.

37. Strauss, H.: Zur Diagnose und Therapie der cardiospastischen Speiseröhrenerweiterung, Berl. klin. Wchnschr. **41**:1261, 1904.

of this dilatation. In 1897, Jaffé [38] proposed such a procedure, in which the dilated esophagus would be diminished in size by excision of long strips of its wall and closure of the defects. Ten years later, Reisinger [39] reported a case in which this was done. He exposed the esophagus through a posterior mediastinotomy opening and attempted to perform esophagoplication, but because the patient went into shock it was necessary to discontinue the operation. However, at a subsequent stage, several weeks later, he was able to excise several strips 2 to 3 cm. in width and 15 cm. in length from the esophageal wall, after which he sutured the defects. Because of subsequent disruption of the esophageal wounds several operations were required before complete healing occurred. However, the patient finally recovered, and the condition was improved.

With a similar rationale, i. e., artificial reduction in the circumference of the esophagus, Meyer,[40] in 1911, performed esophagoplication. Using a transpleural approach through a Schede incision, he exposed the esophagus and sutured together, with interrupted silk sutures, two longitudinal folds of the esophageal wall. A vagolysis was also done. The patient subsequently had an esophageal fistula and died of suppurative mediastinitis. Because of this, Meyer advised the use of catgut instead of silk as suture material for the esophagus. In subsequent reports Meyer [41] reported 2 cases of similar treatment, with poor results, and came to the conclusion that a plastic procedure similar to the Heinicke-Mikulicz pyloroplasty should be done. Whereas these procedures attempted correction of the abnormality by an artificial reduction in the circumference of the esophagus, the rationale of the Freeman [42] procedure was based on diminution in length. Through a cervical incision, Freeman pulled upward on the dilated esophagus until the portion below was "rendered quite taut" and then invaginated the upper segment into the lower without opening the lumen. This intussusception was maintained with a few catgut sutures. The patient remained improved for twenty years.

---

38. Jaffé, K.: Ueber idiopathische Oesophaguserweiterung, München. med. Wchnschr. **44**:386, 1897.

39. Reisinger: Ueber die operative Behandlung der Erweiterung des Oesophagus, Verhandl. d. deutsch. Gesellsch. f. Chir. **36**:86, 1907.

40. Meyer, W.: Impermeable Cardiospasm Successfully Treated by Thoracotomy and Oesophagoplication, Ann. Surg. **53**:293, 1911; Impermeable Cardiospasm Successfully Treated by Thoracotomy and Esophagoplication, J. A. M. A. **56**:1437 (May 20) 1911; Thoracotomy for Impermeable Cardiospasm (Two Cases), with Remarks on Drainage After Intrathoracic Operations, Ann. Surg. **55**:326, 1912.

41. Meyer, W.: (*a*) Operative Treatment of Intractable Cardiospasm, Am. J. Surg. **26**:193, 1912; (*b*) in discussion on Lambert.[82]

42. Freeman, L.: An Operation for Relief of Cardiospasm Associated with Dilatation and Tortuosity of the Esophagus, Tr. Am. S. A. **41**:19, 1923.

The procedure described by Zaaijer [43] in 1912 consisted of gastrostomy at the first stage, esophagostomy being performed subsequently, after several operations in which the esophagus was brought near the chest by preliminary thoracoplasty similar in technic to the operation he employed for cardiac carcinoma. After this, everything the patient swallowed was discharged through the fistula, collected in a bottle and subsequently emptied through the gastrostomy stoma into the stomach.

It is obvious that these procedures attacking the dilated esophagus are as irrational as repair of a hernia by diminishing the size of the sac. At present they may be considered of historical interest only. On the other hand, operative procedures directed at the cardia seem to have a more rational basis, because the obstruction to the passage of food into the stomach appears at this site. Probably because of Mikulicz' influence, the prevalent view at the turn of this century was that a true spasm of the cardiac sphincter exists in this condition. Von Mikulicz drew an analogy between this and spasm of the anal sphincter associated with a fissure. On this basis dilation appeared a rational corrective procedure, and in 1899 Lotheissen [44] suggested the use of "Sondierung ohne Ende," or bougienage without end. This consisted of passing a string orally through the esophagus and out of the stomach through a gastrostomy opening. Retrograde dilation could then be performed by guiding bougies along the string (fig. 1). Von Hacker [45] reported good results from the use of this method.

In 1903, von Mikulicz [46] proposed and performed transgastric dilation of the cardia. Through a laparotomy incision he exposed the stomach and introduced an instrument resembling a glove stretcher through an incision into the anterior gastric wall and into the cardia. "The dilatation was gradually affected to such an extent that the (rubber covered) blades of the instrument were about 7 cm. apart." After this he was able to introduce his second and third fingers through the cardia very readily (fig. 2). After this, over a three month period of observation, the "patient was able to swallow every kind of food without any difficulty." According to Lusena [47] this procedure was performed by Loreta [48] as early as 1884.

---

43. Zaaijer, J. H.: Oesophagotomia thoracalis, Beitr. z. klin. Chir. **77**:497, 1912.

44. Lotheissen, cited by von Hacker and Lotheissen.[1d]

45. von Hacker, in discussion on Reisinger.[39]

46. von Mikulicz, J.: Small Contributions to the Surgery of the Intestinal Tract, Boston M. & S. J. **148**:608, 1903; Zur Pathologie und Therapie des Cardiospasmus, Deutsche med. Wchnschr. **30**:50, 1904; Zur Pathologie und Therapie des Cardiospasmus, ibid. **30**:17, 1904.

47. Lusena, G.: La chirurgia dell'esofago, Cong. Soc. internat. de chir., Rap. **1**: 641, 1932.

48. Loreta, cited by Lusena.[47]

In order to obviate opening of the stomach and possible peritoneal contamination, Rotgans [49] and subsequently others [50] performed dilation of the cardia with the fingers by forcefully invaginating the anterior wall of the stomach up through the cardia. Judd, Vinson and Greenlee [51] suggested the use of a previously swallowed thread as a guide to the cardia when the stomach was opened. Anschütz [52] dilated the cardia with a balloon but opened the abdomen in order to control more accurately the location of the balloon. Von Mikulicz proposed his procedure on the basis that it would permit more accurate control of the dilation

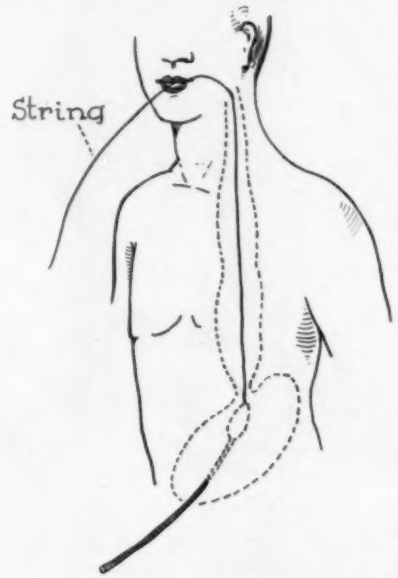


Fig. 1.—Diagrammatic illustration of retrograde dilation in the treatment of achalasia. The bougie is guided along a string introduced orally through the esophagus and out of the stomach through a gastrostomy.

---

49. Rotgans, cited by Zaaijer.[43]

50. (*a*) Wakeley, C. P. G.: A Case of Hiatal Oesophagismus in a Man Aged Thirty-Six Years, Brit. M. J. **1**:589, 1916. (*b*) Schäfer: Operativer Behandlung des Kardiospasmus, Zentralbl. f. Chir. **47**:230, 1920. (*c*) Kümmell, H.: Zur Operation des Kardiospasmus und des Œsophaguscarcinoms, Arch. f. klin. Chir. **117**:193, 1921; (*d*) Zur Operation des Kardiospasmus und des Oesophagus-Carcinoms, Verhandl. d. deutsch. Gesellsch. f. Chir. **45**:327, 1921. (*e*) Gould, G. A.: Dysphagia for Forty-One Years, New England J. Med. **209**:962, 1933. Thieding.[1a]

51. Judd, E. S.; Vinson, P., and Greenlee, D. P.: Retrograde Dilatation of the Oesophagus for Cardiospasm, Surg., Gynec. & Obst. **48**:494, 1929.

52. Anschütz, in discussion on Kümmell,[50d] Verhandl. d. deutsch. Gesellsch. f. Chir. **45**:147, 1921.

and therefore would be less hazardous than blind dilation from above. However, in 1903 he stated that an instrument might be devised to accomplish this result which could be introduced orally. Apparently he was not familiar with Russell's [53] publication. It is obvious that von Mikulicz' procedure accomplishes little more than dilation by the natural oral route and is no less dangerous. Cases have been reported in which the procedure was followed by marked hemorrhage,[54] fatal rupture, mediastinitis [55] and periesophageal abscess.[56] Of the 80 cases

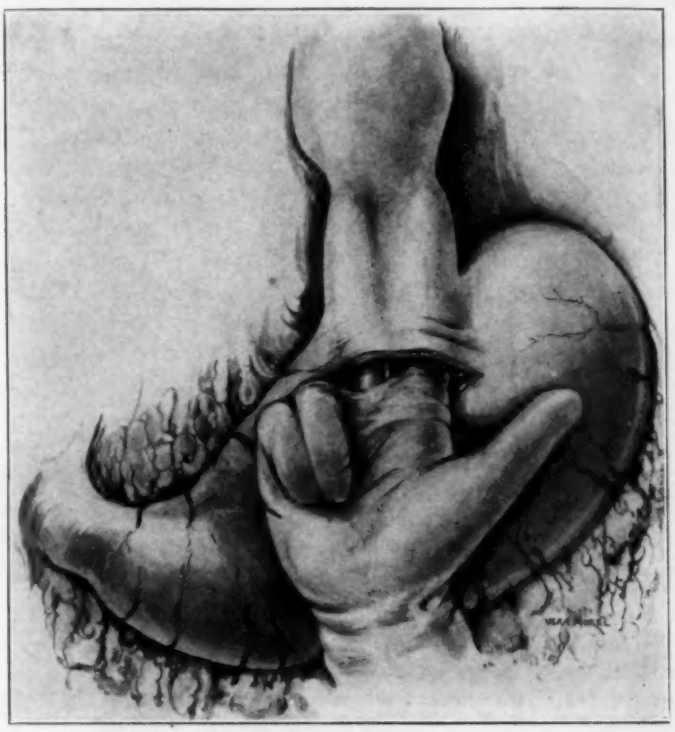


Fig. 2.—Drawing illustrating transgastric dilation of the cardia in the treatment of achalasia.

53. Russell, J. C.: Diagnosis and Treatment of Spasmodic Stricture of the Oesophagus, Brit. M. J. **1**:1450, 1893.

54. Galpern, Y.: Kardiospasmus, Dilatation nach Mikulicz, profuse Blutung, Zentralbl. f. Chir. **57**:87, 1930.

55. Schmilinsky, in discussion on Schäfer.[50b]

56. Gottstein, G.: Die operative Behandlung Cardiospasmus, Zentralbl. f. Chir. **31**:1362, 1904.

collected from the literature,[57] the patients in 7 (8.9 per cent) died and

57. (*a*) Gottstein, G.: Ueber die chirurgische Behandlung des Kardiospasmus, Deutsche med. Wchnschr. **30**:1597, 1904; (*b*) Weitere Fortschritte in der Therapie des chronischen Cardiospasmus, Arch. f. klin. Chir. **87**:497, 1908. (*c*) Ledderhose: Cardiospasmus, Deutsche med. Wchnschr. **30**:1669, 1904; (*d*) in discussion on Gottstein.[56] (*e*) Goldmann: Kardiospasmus, München. med. Wchnschr. **52**:1416, 1905. (*f*) Wilms: Fall von Kardiospasmus mit retrograder Dilatation behandelt, ibid. **52**:144, 1905. (*g*) von Bonsdorff, H.: Contribution to the Question of the Operative Treatment of Cardiospasm, Finska läk.-sällsk. handl. **58**:305, 1906; Nord. tidskr. f. terapi **4**:97, 1906. (*h*) Brünning, F.: Ein Beitrag zur Lehre vom Cardiospasmus, Beitr. z. klin. Chir. **48**:228, 1906. (*i*) Erdmann: Cardiospasm, with Report of an Operative Case, Ann. Surg. **43**:224, 1906. (*j*) Plummer, H. S.: Cardiospasm, with Report of Cases, J. Minnesota M. A. **26**:419, 1906. (*k*) von Eiselsberg, A.: Presentation of Case Before the Medical Society of Vienna, June 21, 1907, Wien. klin. Wchnschr. **20**:811, 1907. (*l*) Graff: Cardiospasmus, Deutsche med. Wchnschr. **33**:447, 1907; (*m*) in discussion on Reisinger.[39] (*n*) Kramer-Petersen: On Cardiospasm, Ugesk. f. læger **52**:1401, 1908. (*o*) Noroth, cited by von Hacker and Lotheissen.[1d] (*p*) Iden, J. H.: Cardiospasm: Report of a Case; Treatment by Operation and Subsequent Dilatation, J. A. M. A. **56**:1438 (May 20) 1911. (*q*) Bowker, G. E.: Discussion of a Note on Cardiospasm, Brit. M. J. **2**:917, 1913. (*r*) Albu: Beitrag zur Lehre von der idiopathischen Oesophagusdilation, München. med. Wchnschr. **64**:749, 1917. (*s*) Schloffer: Kardiospasmus, Wien. klin. Wchnschr. **31**:88, 1918. (*t*) Pamperl, R.: Zur operativen Behandlung des Kardiospasmus, Deutsche Ztschr. f. Chir. **148**:206, 1919. (*u*) Clairmont, P., in discussion on Kümmell,[50d] Verhandl. d. deutsch. Gesellsch. f. Chir. **45**:142, 1921. (*v*) Urrutia, L., cited by von Hacker and Lotheissen.[1d] (*w*) Watts, S. H.: Cardioplasty for Cardiospasm, Tr. Am. S. A. **41**:25, 1923. (*x*) Tuffier: Dilatation de l'œsophage. Méga-œsophage. Opération. Résultat fonctionnel suivi sur radiographie, huit mois après, Bull. et mém. Soc. de chir. de Paris **48**:446, 1922. (*y*) Crone-Münzebrock, E.: Die operative Behandlung des echten Kardiospasmus, Zentralbl. f. Chir. **53**:2386, 1926. (*z*) Doberer, cited by von Hacker and Lotheissen.[1d] (*a'*) Walton, A. J.: The Surgical Treatment of Cardiospasm, Brit. J. Surg. **12**:701, 1925. (*b'*) Enderlen, in discussion on Fromme,[67w] Arch. f. klin. Chir. **157**:138, 1929. (*c'*) Hesse, E.: Ueber chirurgische Eingriffe an der Cardia und am Bauchabschnitt der Speiseröhre bei gutartigen Erkrankungen, Deutsche Ztschr. f. Chir. **213**:23, 1929. (*d'*) Larget, M., and Lamare, J. P.: Cardiospasme ou rétrécissement œsophagien; dilatation forcée du cardia par voie transgastrique; résultats éloignés de deux cas, Bull. et mém. Soc. nat. de chir. **55**:886, 1929. (*e'*) Maconie, A. C.: Cardiospasm Treated Successfully by Operation, Brit. M. J. **1**:398, 1929. (*f'*) Adams, A. W.: Thoracic Stomachs: A Study of Pharyngeal Pouch and Cardiospasm, with Report of Three Cases, ibid. **2**:208, 1930. (*g'*) Lamson, O. F.: Surgical Treatment of Cardiospasm, Northwest Med. **29**:125, 1930. (*h'*) Butler, H. B.: A Case of Achalasia of the Cardia, Brit. M. J. **2**:565, 1931. (*i'*) Jirasek, A.: Un cas d'œsophago-gastro-anastomose réussie, Bull. et mém. Soc. nat. de chir. **57**:1189, 1931. (*j'*) Rieder, W.: Die Therapie des sogenannten Kardiospasmus, Beitr. z. klin. Chir. **151**:495, 1931. (*k'*) Khodkov, V. M.: Surgical Therapy in Connection with Three Cases of Cardiospasm, Sovet. khir. **9**:447, 1936. (*l'*) Gray, H. K., and Skinner, I. C.: The Operative Treatment of Cardiospasm, J. Thoracic Surg., to be published. (*m'*) von Mikulicz.[46] (*n'*) Lusena.[47] (*o'*) Loreta.[48] (*p'*)

(Footnote continued on next page)

the operations in 8 (10.1 per cent) were failures. Good results were reported in 56 (70.8 per cent) and improvement in 6, and the results were unknown in 2.

The next type of surgical procedure directed at the cardia was plastic. With a rationale similar to that of the Ramstedt [58] procedure for hypertrophic pyloric stenosis, Heller,[59] in 1913, performed an extramucous cardiomyotomy which had been proposed by Gottstein [60] in 1901. This

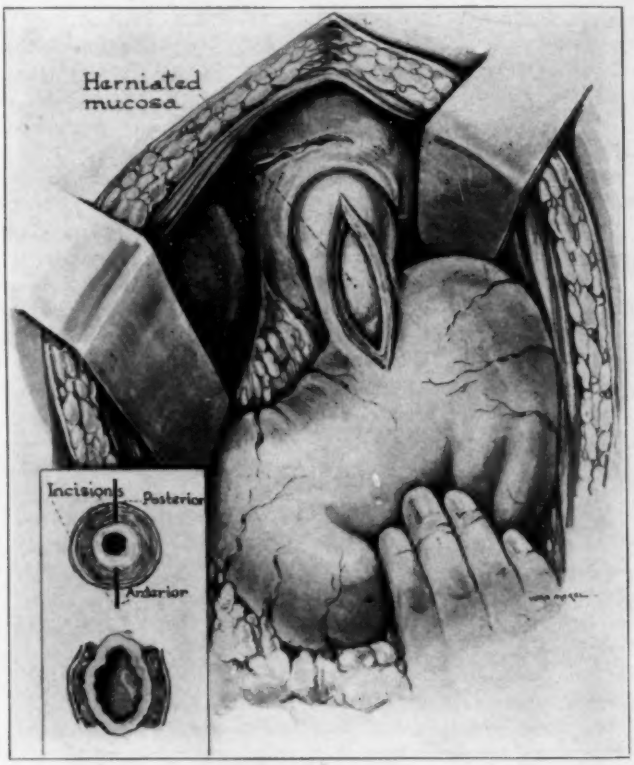


Fig. 3.—Extramucous cardiomyotomy in the treatment of achalasia. The musculature of the cardiac end of the esophagus is incised longitudinally down to the mucosa for about 8 to 10 cm. As is shown in the inset, the vertical incisions are made anteriorly and posteriorly.

Rotgans.[49] (*q'*) Footnote 50. (*r'*) Judd and others.[51] (*s'*) Anschütz.[52] (*t'*) Russell.[53] (*u'*) Galpern.[54] (*v'*) Schmilinsky.[55] (*w'*) Gottstein.[56]

58. Ramstedt, C.: Zur Operation der angeborenen Pylorusstenose, Monatschr. f. Kinderh. **11**:409, 1912.

59. Heller, E.: Extramuköse Cardiaplastik beim chronischen Cardiospasmus mit Dilatation des Oesophagus, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **27**:141, 1913-1914.

60. Gottstein, G.: Technik und Klinik der Oesophagoskopie, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **8**:57, 511 and 99, 1901.

operation consisted of exposure of the stomach and the cardia through a laparotomy incision, mobilization of the cardia and esophagus for 8 to 10 cm. and longitudinal incision of the musculature of the cardiac end of the esophagus down to the mucosa (fig. 3). A vertical incision approximately 8 cm. long was made anteriorly and a similar one posteriorly. Heller [61] insisted on the necessity of two incisions because of the probably continued function of the sphincter due to bridging of the defect by scar if a single incision were made. However, Zaaijer [62] and de Brüne Groeneveldt [63] stated that one incision is sufficient. As usual, this procedure was subsequently modified by a number of surgeons. In 1914, Röpke [64] suggested incising the hiatus to permit better mobilization and removing the periesophageal tissues down to the mucosa for a distance of 6 cm. Schaldemose [65] preferred making a circular incision over a grooved director inserted through a small opening down to the mucosa and then up between the mucosa and the muscle layer. Similarly, Oliveira Mattos [66] suggested that a forceps be used to dissect between the mucosa and the muscle layer before the operator incises the latter and resects a part of it. These modifications have the obvious purpose of avoiding opening the lumen by accidentally tearing or incising the mucosa. That this is not merely a theoretic objection is shown by the fact that of 104 collected cases,[67] accidental tearing of the mucosa

61. Heller, in (*a*) discussion on Röpke;[64a] (*b*) in discussion on Kümmell,[50d] Verhandl. d. deutsch. Gesellsch. f. Chir. **45**:144, 1921; (*c*) Die Behandlung des Kardiospasmus, Med. Welt **6**:1675, 1932.

62. Zaaijer, J. H.: Cardiospasm in the Aged, Ann. Surg. **77**:615, 1923.

63. de Brüne Groeneveldt, J. R.: On Cardiospasm, Nederl. tijdschr. v. geneesk. **2**:1281, 1918.

64. Röpke: (*a*) Zur Operation des Oesophagospasmus, Verhandl. d. deutsch. Gesellsch. f. Chir. **43**:121, 1914; (*b*) in discussion on Kümmell,[50d] ibid. **45**:146, 1921; (*c*) in discussion on Fromme,[67w] Arch. f. klin. Chir. **157**:136, 1929.

65. Schaldemose, V.: Operated Cases of Idiopathic Dilatation of the Esophagus, Nord. kir. för. forhandl. **11**:231, 1916.

66. Oliviera Mattos, J.: Tratamentodo megaesofago (acalasia do cardia) pela esfíncterectomia cardica, Rev. Assoc. paulista de med. **13**:217, 1938.

67. (*a*) Ambrumjanz: Ueber einer Modification der Hellerschen Operation beim Kardiospasmus, Zentralorg. f. d. ges. Chir. u. Grenzgeb. **50**:747, 1930. (*b*) Soupault, R., and Hamburger, M.: Rétrécissement oesophagocardiaque dit essentiel opéré et guéri, Arch. d. mal. de l'app. digestif **22**:48, 1932; (*c*) Résultat éloigné d'une œsophago-cardiotomie pour rétrécissement œsophagien, ibid. **23**:103, 1933; (*d*) Les résultats éloignes de l'operation de l'œsophago-cardiotomie extramuqueuse, ibid. **26**:942, 1936. (*e*) Borchgrevink, O., in discussion on Schaldemose;[65] (*f*) in discussion on Kümmell,[50d] Verhandl. d. deutsch. Gesellsch. f. Chir. **45**: 148, 1921. (*g*) Exalt, O. J.: A Case of Chronic Cardiospasm, Nederl. tijdschr. v. geneesk. **2**:1276, 1918. (*h*) Lindström, T. E.: A Case of Cardiospasm with Dilatation of the Esophagus, Hygiea **80**:1169, 1918. (*i*) Noordenbos, W.: Cardiospasmus, Nederl. tijdschr. v. geneesk. **2**:1278, 1918. (*j*) Zaaijer, J. H.:

(Footnote continued on next page)

occurred in 14, and in 2 of these the patients died of consequent infection. There were a third death, due to perforation following bougienage after the completion of the operation, and a fourth, due to pulmonary embolism, giving a mortality rate of 4 per cent. Recurrence took place in 14 cases of the collected group. The results were considered good in 80 and improvement was observed in 6 others.

Kardiospasm und Esophagusaffection, Thesis, Leyden, 1918; abstracted, Zentralbl. f. Chir. **46**:57, 1919; (*k*) On Surgery of the Oesophagus, Cong. Soc. internat. de chir., Rap. **1**:485, 1932. (*l*) Mintz, W.: Operative Eingriffe bei Kardiospasmus und Megaösophagie, Deutsche med. Wchnschr. **46**:1296, 1920. (*m*) Enderlen, in discussion on Kümmell,[50d] Verhandl. d. deutsch. Gesellsch. f. Chir. **45**:147, 1921. (*n*) Payr, E., cited by Heller;[61b] (*o*) in discussion on Kümmell,[50d] ibid. **45**:147, 1921; (*p*) Chirurgische Behandlung des Kardiospasmus, Zentralbl. f. Chir. **56**: 3103, 1929. (*q*) König, cited by von Hacker and Lotheissen.[1d] (*r*) Hörhammer, C.: Das Einmanschettierungsverfahren nach Goepel an den kardialen Magenpartien, mit besonderer Berücksichtigung Einpflanzung des Oesophagus, Zentralbl. f. Chir. **50**:633, 1923. (*s*) Hurst, A. F., and Rowlands, R. P.: Case of Achalasia of Cardia Relieved by Operation, Proc. Roy. Soc. Med. (Clin. Sect.) **17**:45, 1924. (*t*) Reise: Kardiospasmus, Zentralbl. f. Chir. **51**:2137, 1924. (*u*) Camargo, A. C.; Vampré, E., and Parisi, R.: Mal de engasgo; cura pela operacão de Heller; 2 observacão, Bol. Soc. de med. e cir. de São Paulo **7**:54, 1924-1925. (*v*) Chaves, J. A.: Therapeutica cirúrgica do mal de engasgo, Brasil-med. **41**:43, 1927. (*w*) Fromme, A.: Erfahrungen mit der Operation des Kardiospasmus, Arch. f. klin. Chir. **157**:606, 1929. (*x*) Delbet, P.: Mégacesophage. Opération par voie abdominale, Bull. et mém. Soc. nat. de chir. **55**:481, 1929; (*y*) Troisième cas de cardiospasme opéré par voie abdominale, ibid. **57**:586 and 1056, 1931; (*z*) Rétrécissement simple de l'œsophage (cardiospasme) opéré par la voie abdominale, ibid. **57**:1057, 1931. (*a'*) Oberthür, H.: Traitement chirurgical de la dilatation dite idiopathique de l'œsophage, Arch. d. mal. d. l'app. digestif **21**:649, 1931. (*b'*) Jacobovici, I., and Hanganut: La cardio-myotonie dans le traitement de la dilatation idiopathique de l'œsophage, Romania med. **9**:325, 1931. (*c'*) Turner, G. G.: Some Experiences in the Surgery of the Esophagus, New England J. Med. **205**:657, 1931; (*d'*) Personal Experience in the Surgery of the Lower Esophagus, Cong. Soc. internat. de chir., Rap. **1**:725, 1932. (*e'*) Charbonnel and Massé: Le traitement chirurgical du mégacœsophage. L'œsophago-cardiomyotomie extramuqueuse (Un cas personnel), Bordeaux chir. **3**:213, 1932; (*f'*) Méga-œsophage avec rétrécissement inférieur; opération par voie abdominale (cardiœesophagotomie extramuqueuse). Résultat datant de sept mois, Bull. et mém. Soc. nat. de chir. **58**:1092, 1932. (*g'*) Charbonnel, in discussion on Chenut.[95e] (*h'*) Jáuregui, P., and Subiza, V.: Cardioespasmo operado por vía abdominal. Curacíon, Bol. y trab., Soc. de cir. de Buenos Aires **16**:1525, 1932. (*i'*) Soupault, R.: L'œsophago-cardiotomie dans les rétrécissements simples de l'œsophage inférieur, Cong. Soc. internat. de chir., Rap. **1**:759, 1932; (*j'*) L'œsophago-cardiotomie extra-muqueuse (opération de Heller), J. de chir. **41**:727, 1933; (*k'*) Les rétrécissements cardio-œsophagiens (ex-cardiospasmes), Médecine **15**:889, 1934. (*l'*) Hartung, in discussion on Fromme.[91q] (*m'*) Recalde, J. F.: Cardiospasmo, disfagio y mega-esófago; simpaticectomía esofágica; estadística quirúrgica, Semana méd. **1**:1552, 1933. (*n'*) Vampré, E.: Le "mal de engasgo" et son traitement chirurgical, Rev. sud-am. de méd. et de chir. **4**:493, 1933. (*o'*) Carayannopoulos, A.: Sur un cas

The other plastic operation on the cardia is analogous to the Heinecke-Mikulicz pyloroplasty.[68] This was first performed by Wendel,[69]

---

de mégacesophage, Bull. et mém. Soc. nat. de chir. **60**:14, 1934. (*p'*) Fruchaud, H.: Un cas de rétrécissement cardio-œsophagien essentiel traité par opération de Heller, ibid. **60**:1264, 1934. (*q'*) Jauve: Sténose cardio-œsophagienne chez l'enfant. Opération de Heller, Lyon chir. **31**:586, 1934. (*r'*) Leriche, R., in discussion on Jauve.[67q'] (*s'*) Rohde, C.: Zur Operation des Kardiospasmus, Zentralbl. f. Chir. **61**:977, 1934. (*t'*) Buzoianu, G., and Ionescu, D.: Cardiomyotomy in Esophageal Cardiospasm, Rev. de chir., Bucureşti **38**:137, 1935. (*u'*) Diez, J.: Cardioespasmo y operación de Heyrovsky, Bol. y trab., Soc. de cir. de Buenos Aires **19**:924, 1935; (*v'*) Cardioesopasmo y operación de Heller, ibid. **19**:846, 1935; (*w'*) El tratamiento quirúrgico del cardioespasmo (operaciones de Heller y de Heyrovsky), Prensa méd. argent. **23**:155, 1936. (*x'*) Lotheissen, G.: Kardiendyse, die neue Operation zur Behandlung des Kardiospasmus, Zentralbl. f. Chir. **62**:2658, 1935. (*y'*) Baumgartner, in discussion on Magnant.[67b''] (*z'*) Hamant, A., and Chalnot: Deux cas de mégacœsophages traités par la technique de Heller, Rev. méd. de Nancy **64**:294, 1936. (*a''*) Küss, G.: A propos des rétrécissements cardio-œsophagiens, Mém. Acad. de chir. **62**:838, 1936. (*b''*) Magnant, J. S.: Rétrécissement cardio-œsophagien. Intervention par voie abdominale. Résultat éloigné, ibid. **62**: 761, 1936. (*c''*) Marinacci, S.: Esofago-cardiotomia extramucosa (operazione di Heller), Arch. ital. di chir. **44**:698, 1936. (*d''*) Most, in discussion on Frey,[80b] Arch. f. klin. Chir. **186**:19, 1936. (*e''*) Pieri, G.: Sulla cura chirurgica del cardiospasmo, Riforma med. **52**:1051, 1936. (*f''*) Desplas, B., and Aimé, P.: Deux cas de sténos hypertrophique du cardia, Mém. Acad. de chir. **62**:843, 1936. (*g''*) Desplas, B.; Durand, G., and Aimé, P.: Mégacœsophage par sténose suscardiaque, cravate musculaire et cercle vasculaire anormal sténosant. Intervention. Guérison. Résultats radiographiques, Arch. d. mal. de l'app. digestif **27**:877, 1937. (*h''*) Abbeloos: Observation nouvelle de cardiospasme avec mégacœsophage guéri par opération de Heller, J. de chir. et ann. Soc. belge de chir. **36-34**:631, 1937. (*i''*) Girard, L., and Ranjard, C.: Phréno-cardiospasme et cardiotomie, Oto-rhino-laryng. internat. **21**:132, 1937. (*j''*) Vasconcelos, E., and Botelho, G.: Cirurgia do megaesôfago, São Paulo, Coimpanhia Editora Nacional, 1937. (*k''*) Huard, P., and Long, M.: Deux cas de rétrécissement cardio-œsophagien traités par l'opération de Heller, Rev. méd. franç. d'Extrême-Orient **16**:31, 1938. (*l''*) Rousseaux: Cardiospasme chronique. Opération de Heller. Résultat éloigné, Rev. méd. de Nancy **67**:28, 1939. (*m''*) Christide, E.: Méga-œsophage opéré par le procéde de "Heller" et guéri, Lyon chir. **36**:690, 1940. (*n''*) Ferrari, R. C., and Pérez Zabala, M.: La operación de Heller en el tratamiento de los fenómenos de estenosis benignos al nivel del cardias, Bol. Inst. de clin. quir. **12**:64, 1936; (*o''*) La operación de Heller en el tratamiento del llamado cardiospasmo, Semana méd. **1**:1905, 1936. (*p''*) Pérez Zabala, M., and Ferrari, R. C.: La operación de Heller en el tratamiento de los fenómenos de estenosis benignos al nivel del cardias, Bol. y trab., Soc. de cir. de Buenos Aires **20**:176, 1936. (*q''*) Scrimger.[8e] (*r''*) Clairmont.[57u] (*s''*) Jirasek.[57l'] (*t''*) Heller.[59] (*u''*) Heller.[61] (*v''*) Zaaijer.[62] (*w''*) de Brüne Groeneveldt.[63] (*x''*) Röpke.[64] (*y''*) Schaldemose.[65]

68. Fronmüller, F.: Operation der Pylorusstenose, Inaug. Dissert., Fürth, A. Schröder, 1886. von Mikulicz, J.: Zur operativen Behandlung des stenosirenden Magengeschwüres, Arch. f. klin. Chir. **37**:79, 1888.

69. Wendel, W.: Zur Chirurgie des Oesophagus, Arch. f. klin. Chir. **93**:311, 1910.

in 1910, although it had been suggested by Marwedel[70] seven years previously. The procedure consists of making a longitudinal incision through the entire wall of the cardia and suturing the defect transversely, thus increasing the diameter of the lumen at this site (fig. 4). In the United States, Meyer,[41b] in 1913, successfully performed this operation through a transpleural approach. Since then there have been 20 cases[71] reported, with 1 death.[71h] Good results were obtained in

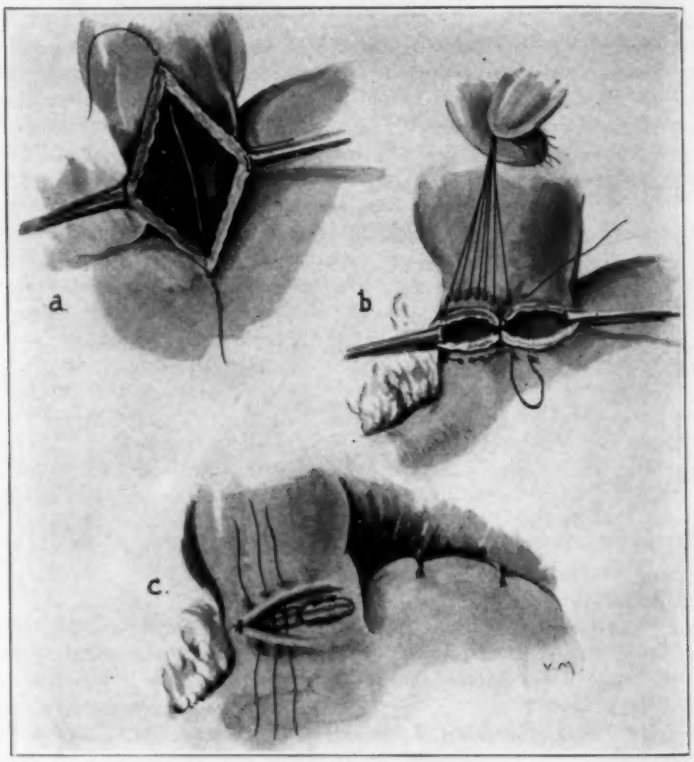


Fig. 4.—Cardioplasty in the treatment of achalasia. A longitudinal incision is made (*a*) through the entire wall of the cardia, and the defect is sutured transversely (*b* and *c*).

---

70. Marwedel, G.: Die Aufklappung des Rippenbogens zur Erleichterung operativer Eingriffe im Hypochondrium und im Zewerchfellkuppelraum, Zentralbl. f. Chir. **30**:938, 1903.

71. (*a*) Lecène, P.: Sténose inflammatoire avec spasme du cardia; dilatation œsophagienne considérable. Cardioplastie. Guérison, Bull. et mém. Soc. de chir. de Paris **45**:710, 1919; (*b*) in discussion on Larget and Lamare.[57d'] (*c*) Sencert, L., and Simon, R.: Le traitement opératoire de la dilatation idiopathique de

all except 1 case. This was reported by Bull,[1b] who apparently intended to perform the operation of Heller but, because the mucosa was accidentally torn, sutured the defect transversely. The patient had a recurrence, and an esophagogastrostomy was subsequently done. A modification of this procedure, analogous to the Fredet-Weber type of pyloroplasty,[72] was proposed by Girard [73] in 1915. In order to obviate opening the lumen and thus avoid possible peritoneal contamination, Girard performed the operation by making a longitudinal incision down to the mucosa and then suturing the resultant defect transversely (fig. 5). He reported 2 cases with successful results. In 1933, Ohsawa [74] reported 6 cases in which operation was similarly performed through an approach designated as a free transdiaphragmatic thoracolaparotomy, with good results in all. We have been able to collect 14 cases [75] in which the

---

l'œsophage, Rev. de chir. de Paris **59**:355, 1921. (*d*) Grégoire, R.: Voie d'accès sur le segment cardio-œsophage permettant d'éviter la plèvre et le péritoine, J. de chir. **21**:673, 1923; (*e*) Méthode permettant d'atteindre le segment cardio-œsophage en évitant la plèvre et le péritoine, Bull. et mém. Soc. de chir. de Paris **49**:600, 1923; (*f*) Résultat au bout d'un an d'une intervention pour dilatation idiopathique de l'oesophage. Section du diaphragme par voie thoraco abdominale extra-séreuse. Œsophago-gastroplastie, Bull. et mém. Soc. nat. de chir. **49**:1322, 1923; (*g*) A propos de la dilatation idiopathique de l'œsophage, ibid. **49**:1502, 1923; (*h*) Pathogénie et traitement de la dilatation idiopathique de l'œsophage, Arch. d. mal. de l'app. digestif **14**:455, 1924; (*i*) Phrénospasme. Œsophago-gastroplastie par voie thoraco-abdominale extra-sérause, Bull. et mém. Soc. nat. de chir. **52**:757, 1926; (*j*) Etat actuel de la chirurgie de l'œsophage, Cong. Soc. internat. de chir., Rap. **1**:219, 1932. (*k*) Lagas, F. M., cited by Charbonnel and Massé.[67t'] (*l*) Wendel, in discussion on Rieder.[57j'] (*m*) Graham, R., cited by Janes.[81] (*n*) Murray, G., cited by Janes.[81] (*o*) Bull.[1b] (*p*) Janes.[81] (*q*) Footnotes 57 *c'* and *k'*. (*r*) Footnotes 67 *u* and *k'*.

72. Fredet: La sténose hypertrophique du pylore chez le nouveau-né, Arch. d. mal. de l'app. digestif **2**:393, 1908. Weber, W.: Ueber eine technische Neuerung bei der Operation der Pylorus-Stenose des Säuglings, Berl. klin. Wchnschr. **47**: 763, 1910.

73. Girard, cited by von Hacker and Lotheissen.[1d]

74. Ohsawa, T.: The Surgery of the Esophagus, Arch. f. jap. Chir. **10**:605, 1933.

75. (*a*) Bard, L.: Le mégacesophage, cas particulier des dilatations idiopathiques des organes cavitaires, Arch. d. mal. de l'app. digestif **9**:541, 1918; (*b*) Note complémentaire sur le mégacesophage, ibid. **10**:116, 1919. (*c*) Hertz, J., and Braine, J.: Phréno-spasme. Elargissement de l'orifice diaphragmatique de l'œsophage. Œsophagoplastie par voie thoraco-abdominale extraséreuse, Bull. et mém. Soc. nat. de chir. **50**:569, 1924. (*d*) Braine, J.: Accès de l'œsophage médiastinal inférieur et du cardia par la voie thoraco-abdominale postérieure sous-pleuro-péritonéale spécialement dans les dilatations "idiopathiques" de l'œsophage, mégacesophages et phrénospasmes, Cong. Soc. internat. de chir., Rap. **1**:765, 1932. (*e*) Lorenzo, R., and Boto, D.: Megaesófago de larga data; éxito quirúrgico, Prensa méd. argent. **24**:349, 1937. (*f*) Fromme.[67w] (*g*) Girard.[73] (*h*) Ohsawa.[74]

extramucous cardioplasty was done. There were no deaths, and good results were obtained in all. Thus, in 36 cases in which cardioplasty was done there was only 1 death, and only 1 recurrence was reported. Good results were obtained in 93.1 per cent of cases. Such impressive results would not be expected to follow a procedure which obviously increased the diameter of the cardia relatively little.

Excision of the cardia followed by esophagogastrostomy was originally proposed by Rumpel [76] in 1897 and was performed by Bier in 1920.

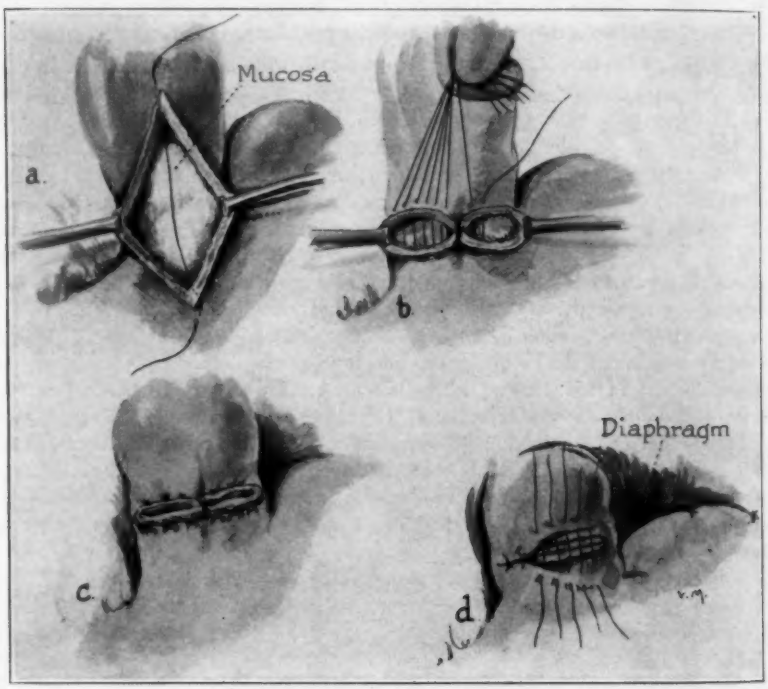


Fig. 5.—Extramucous cardioplasty in the treatment of achalasia. A longitudinal incision is made through the musculature of the cardiac end of the esophagus down to the mucosa, and the defect is sutured transversely.

Bier's case was reported by Pribram,[77] who did not consider the condition typical of cardiospasm, because there was no hypertrophy at the

76. Rumpel, T.: Die klinische Diagnose der spindelförmigen Speiseröhrenerweiterung, München. med. Wchnschr. **44**:383, 1897.

77. Pribram, B. O.: Zur Pathologie und Chirurgie der spastischen Neurosen, Arch. f. klin. chir. **120**:207, 1920; in discussion on Kümmell,[50d] Verhandl. d. deutsch. Gesellsch. f. Chir. **45**:147, 1921.

lower end of the esophagus and the cardia was wide open. However, the clinical manifestations were characteristic of cardiospasm. The patient died of cardiac failure the day following the operation. A second case, in which treatment was successful, was reported more recently by Radlinski.[78] The fact that these are the only 2 cases that have been reported emphasizes the objectionable features of the opera-

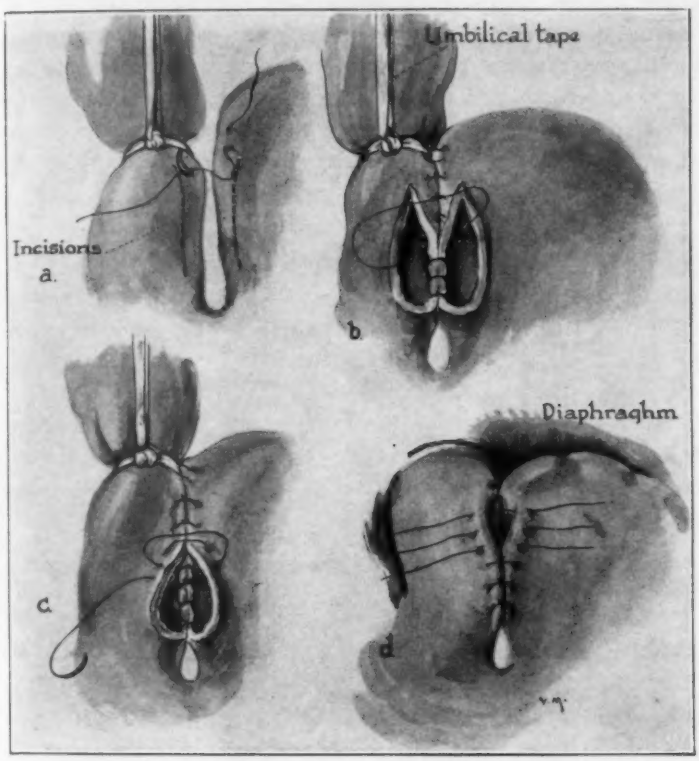


Fig. 6.—Technic of side to side esophagogastrostomy as originally done by Heyrovsky in the treatment of achalasia. A new opening is made between the esophagus and the stomach, thus short-circuiting the cardiac orifice.

tion. Such a radical procedure, in our opinion, is justified only in the presence of a malignant growth.

Of the various procedures directed at the cardia, probably the most rational is esophagogastrostomy. This was first performed successfully

78. Radlinski: Observations de chirurgie œsophagienne, Bruxelles-méd. **17**:202, 1936.

in a case of achalasia by Heyrovsky,[16b] in 1912. Gosset,[79] in 1903, had suggested this procedure for strictures of the cardia and had demonstrated its feasibility in cadavers and dogs, using a transpleural approach. The operation as performed by Heyrovsky consists of exposure of the stomach through a laparotomy incision and mobilization of the cardia and esophagus for a distance of about 8 cm., followed by a side to side anastomosis between the esophagus and the stomach, short-circuiting the cardiac orifice. Because, as was emphasized by Frey,[80] in this type of procedure a spur remains at the cardia which may cause some obstruction to the free passage of ingested material from the esophagus into the stomach, Gröndahl,[81] in 1916, modified the operation so that the anastomosis is made similar to that of the Finney gastroduodenostomy. In this way the cardiac spur is destroyed and a much wider opening

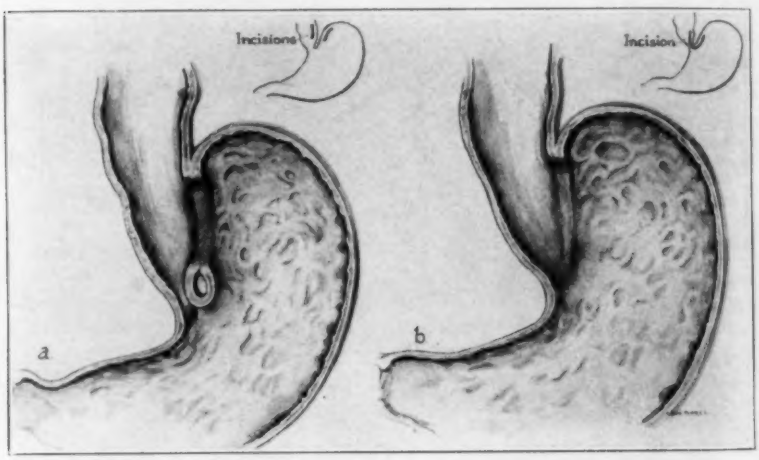


Fig. 7.—Differences in (*a*) the original Heyrovsky side to side esophagogastrostomy and (*b*) Grondahl's modification. Whereas in *a* a spur remains at the cardia which may cause some obstruction to the free passage of ingested material from the esophagus into the stomach, in *b* the cardiac spur is destroyed and a much wider opening is created between the esophagus and stomach.

---

79. Gosset, A.: De l'œsophago-gastrostomie transdiaphragmatique opération de Biondi, Rev. de chir. **28**:694, 1903.

80. (*a*) Frey, E. K.: Zur Technik der Osophagogastrostomie, Zentralbl. f. Chir. **59**:845, 1932; (*b*) Zur Behandlung des Kardiospasmus, Arch. f. klin. Chir. **186**:466, 1936. (*c*) Frey, E. K., and Duschl, L.: Der Kardiospasmus, München. med. Wchnschr. **84**:1374, 1937. (*d*) Frey, E. K.: Die cardioplastische Oesophago-Gastrostomie, Zentralbl. f. Chir. **65**:2, 1938.

81. Gröndahl, N. B.: Plastic Operation for Cardiospasm, Nord. kir. för. förhandl. **11**:236, 1916.

is created between the esophagus and the stomach (fig. 7). In the United States, Lambert,[82] in 1913, achieved the same end by crushing the spur with a forceps. After the cardia and the esophagus were exposed, a large crushing forceps was introduced into the stomach through a gastrostomy opening and applied in such a way that one blade was inserted through the cardia into the esophagus and the other into the fundus of the stomach. The clamp was closed, and a few interrupted sutures were used to approximate the esophagus and the stomach about it. On the eighth postoperative day the spur had been crushed, and the clamp was removed. Instead of a crushing clamp, Keller [83] used a heavy fish line to cut through the spur.

Because this procedure of esophagogastrostomy is considered the most rational, a fuller description of the operation is deemed justifiable. Whereas some surgeons [84] prefer the transpleural supradiaphragmatic approach, others [85] consider the abdominal approach better. In our opinion the abdominal approach is more desirable, because the peritoneum is more resistant to infection than is the pleura, and the exposure is just as satisfactory. As has been emphasized by Lambert [82a] and others,[86] division of the left lateral hepatic ligament should be done in order to permit retraction of the left lobe of the liver and better exposure of the cardia (fig. 8*a*). The peritoneum over the esophagus is circumcised at the site of its reflection on the diaphragm, and by sharp and blunt dissection the esophagus is freed circumferentially (fig. 8*b*). A

82. Lambert, A. V. S.: (*a*) Oesophago-Gastrostomy for Cardiospasm, Ann. Surg. **58**:415, 1913; (*b*) Treatment of Diffuse Dilatation of the Oesophagus by Operation: Description of a Hitherto Unpublished Method; Report of a Case, Surg., Gynec. & Obst. **18**:1, 1914.

83. Keller, W. L.: Operative Relief of Cardiospasm Where Dilatation Has Failed, Ann. Surg. **88**:58, 1928.

84. (*a*) Sauerbruch, F., in discussion on Kümmell,[50d] Verhandl. d. deutsch. Gesellsch. f. Chir. **45**:149, 1921; (*b*) Der Kardiospasmus, in Chirurgie der Brustorgane, Berlin, Julius Springer, 1925, vol. 2, p. 572. (*c*) Sauerbruch, F., and O'Shaughnessey, L.: Thoracic Surgery, Baltimore, William Wood & Company, 1937. (*d*) Breitner, B.: Oesophagogastrostomie wegen Kardiospasmus, Wien. med. Wchnschr. **78**:765, 1928; (*e*) Ein durch Esophagogastrostomie geheilter Fall von Kardiospasmus, ibid. **79**:508, 1929; (*f*) Bericht über einen weiteren Fall von Oesophago-Gastrostomie, ibid. **80**:90, 1930. (*g*) Denk, W., in discussion on Schnitzler.[91e] (*h*) Enderlen: Demonstration eines Präparates von Magenoesophagusanastomose, Zentralbl. f. Chir. **40**:92, 1913. Frey.[80]

85. Oberthür.[67a'] Turner.[67e'] Turner.[67d'] Lambert.[82a]

86. (*a*) Prat, D.: Deux cas de mégacesophage, Bull. et mém. Soc. nat. de chir. **50**:519, 1924. (*b*) Herzberg, B.: Die Anatomie des Bauchabschnittes der Speiseröhre, Deutsche Ztschr. f. Chir. **242**:265, 1934; (*c*) Vergleichende Darstellung verschiedener Methoden operativer Freilegung des Bauchabschnittes der Speiseröhre im Lichte anatomischer und klinischer Untersuchungen, ibid. **242**:290, 1934. Footnotes 67 s, x, y, z, a', c' and d'.

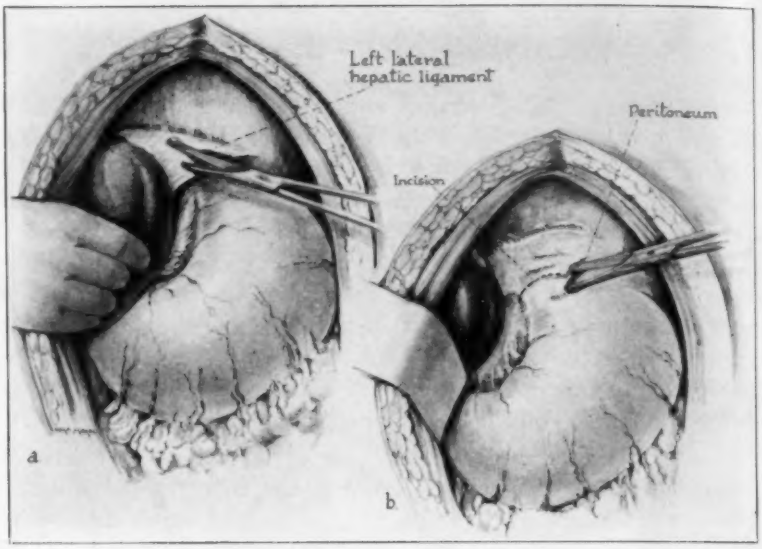


Fig. 8.—Technic of esophagogastrostomy in the treatment of achalasia. Through an abdominal incision the left lateral hepatic ligament is divided in order to permit retraction of the left lobe of the liver and better exposure of the cardia (*a*). The peritoneum over the esophagus is circumcised at the site of its reflection on the diaphragm, and by sharp and blunt dissection the esophagus is freed circumferentially (*b*).

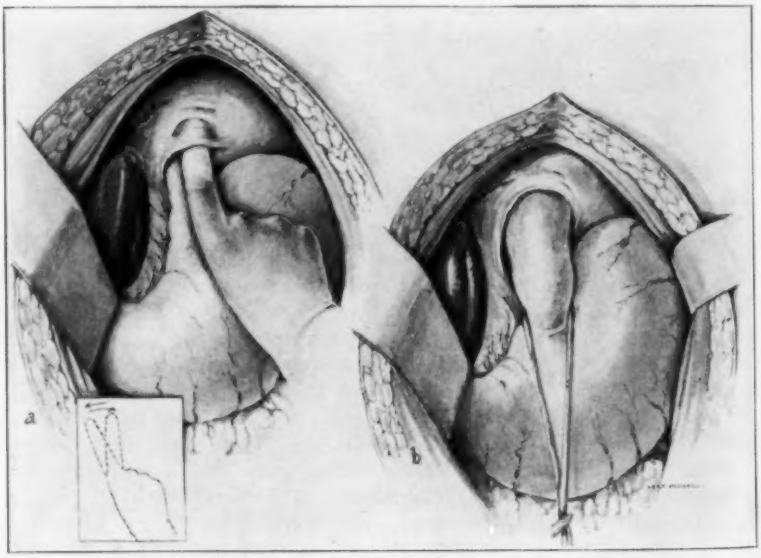


Fig. 9.—Continuation of the procedure shown in figure 8. The esophagus is carefully mobilized (*a*) by blunt dissection with the index finger, with the motions shown in the inset. Traction on a sling of umbilical tape placed around the esophagus aids in its mobilization (*b*) as it is gradually pulled down into the peritoneal cavity for approximately 8 to 10 cm.

sling of umbilical tape is then placed around it to permit traction downward on the esophagus. By blunt dissection with the index finger the esophagus is mobilized from its hiatus and gradually pulled down into the peritoneal cavity for a distance of approximately 8 to 10 cm. (fig. 9). At the uppermost portion of the mobilized esophagus a strip of umbilical tape is tied tightly around the esophagus, a technical suggestion made

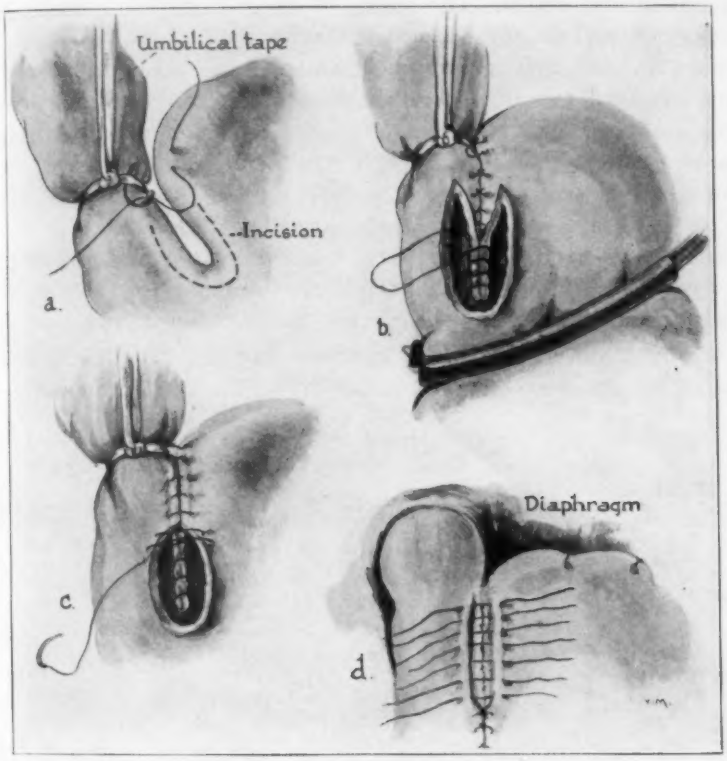


Fig. 10.—Continuation of the procedure described in figure 9. A strip of umbilical tape is tied tightly (*a*) around the uppermost portion of the mobilized esophagus. This serves the purpose of preventing spillage from the esophagus and is used to anchor the fundus of the stomach to the esophagus by placing the anchor suture around the tape and thus to obviate tearing the friable esophageal wall. The dotted line shows the site of the incision through the stomach and the esophagus. A rubber-covered clamp is applied (*b*) to the stomach below the site of anastomosis to prevent spillage. After placing of the first posterior row of interrupted cotton sutures the stomach and esophageal walls are incised as indicated and the second posterior row of sutures is applied with a through and through stitch with no. 00 chromic catgut on an atraumatic needle; then (*c*) this is brought anteriorly as a Connell stitch. A final row of interrupted Lambert cotton sutures is applied (*d*), and a few interrupted sutures are used to tack the diaphragm to the fundus of the stomach.

by Fromme[87] (fig. 10 *a*). This serves two purposes. It prevents spillage from the esophagus above, and it is used to anchor the fundus of the stomach to the esophagus by placing the anchor suture around the tape, thus obviating tearing the friable esophageal wall at a point where there is considerable tension. A posterior row of interrupted quilting cotton sutures[88] is installed (fig. 10 *b*). Paralleling this row of sutures, an incision is made through the stomach and the esophagus and carried through the cardia. The second posterior row of sutures is applied as a through and through stitch with 00 chromic catgut on an atraumatic needle (fig. 10 *b*). This is brought anteriorly as a Connell stitch (fig. 10 *c*). A final row of interrupted Lambert cotton sutures is applied, thus completing the anastomosis (fig. 10 *d*). A few interrupted cotton sutures are used to tack the diaphragm to the esophagus and to the fundus of the stomach in order to avoid traction on the suture line. The piece of tape tied around the esophagus is cut and removed. An omental graft may be used over the anastomosis as suggested by Palugyay.[89] Preliminary paralysis of the right leaf of the diaphragm by phrenicoexeresis or phrenicopraxis has been advocated by some.[90]

We have collected from the literature 88 cases in which esophagogastrostomy has been done for achalasia.[91] There were 5 deaths (6.6

87. Fromme, A.: Ueber Ursachen und Behandlungsmethoden des soggenannten Kardiospasmus auf Grund klinischer Erfahrung, Beitr. z. klin. Chir. **162**:337, 1935.

88. Meade, W., and Ochsner, A.: Spool Cotton as a Suture Material, J. A. M. A. **113**:2230 (Dec. 16) 1939; The Relative Value of Catgut, Silk, Linen and Cotton as Suture Materials, Surgery **7**:485, 1940.

89. Palugyay, J.: Die Oesophago-Gastro-Anastomose nach Heyrovsky im Röntgenbild. Ein Beitrag zum funktionellen Verhalten der Speiseröhre und des Magens nach der Operation, Arch. f. klin. Chir. **125**:554, 1923.

90. Jirasek.[57f] Turner.[67e] Frey.[80] Footnotes 84 *d*, *e* and *f*.

91. (*a*) Demmer, F.: Kardiospasmus, Wien. klin. Wchnschr. **25**:1928, 1912. (*b*) Exner, A.: Beitrag zur subphrenischen Oesophago-gastrotomie, ibid. **30**: 886, 1917. (*c*) Schnitzler, J.: Subphrenische Oesophagogastrostomie, ibid. **31**:768, 1918. (*d*) Finsterer, H., in discussion on Kümmell,[50d] Verhandl. d. deutsch. Gesellsch. f. Chir. **45**:148, 1921; (*e*) Zur Therapie des Kardiospasmus und der Kardiastenose, Wien. klin. Wchnschr. **35**:471, 1922. (*f*) Zaaijer, J. H., cited by Häggstrom.[91m] (*g*) Nystrom, cited by Häggstrom.[91m] (*h*) Gerulanos, cited by Haglund. (*i*) Michaelsson, cited by Häggström.[91m] (*j*) Toole, H.: Ein Fall von hochgradigen Kardiospasmus geheilt durch Oesophagogastrostomie, Arch. f. klin. Chir. **151**:761, 1928. (*k*) Giertz, K. H.: Dilatatio esophagi idiopathica. Svensk. kir. sekt. förh., 1929; cited by Häggström;[91m] (*l*) Operation of Case of Idiopathic Dilatation of the Esophagus **93**:190, 1931. (*m*) Häggström, P.: Zwei Fälle von Kardiospasmus und Osophagus-Dilatation, die mit glücklichen Ausgang nach Heyrovsky operiert wurden, Acta chir. Scandinav. **66**:345, 1930. (*n*) Corrêa Netto, A.: Do tratamento do cardiospasmo pela gastro-esophago-anastomose infradiaphragmatic (operacão

per cent) and only 1 poor result. In addition, we have successfully employed this procedure in 2 cases. An interesting feature in the results of this operation (as well as some of the others) is the observation made by numerous surgeons that functionally the patients are considerably better than the roentgen studies would indicate. Thus, in many cases the esophagus appears still dilated after operation, although the patient is able to swallow normally.

Because one of the theories of achalasia incriminates the diaphragm, it was logical to assume that the condition might be corrected by an operative procedure directed at this structure. In 1914, Bassler [80a] proposed division of the diaphragmatic crura on the assumption that spasm of the muscle fibers was the cause of the obstruction. This was done by Braine and Metivet,[92] with a good immediate result. In France this procedure seemed popular. In 1933, Vampré stated that after performing this operation in 5 or 6 cases with poor results he was forced to the conclusion that it is based on a false premise. In addition to enlarging the diaphragmatic hiatus, a number of surgeons have emphasized the importance of drawing the esophagus down and "straightening it out." Apparently this was first suggested by von Hacker [93] in 1913. Whereas some [94] have stressed the importance of this, apparently they are not fully convinced of its value, as they also perform a cardioplasty. Turner [67c'] stated that he finally discontinued using this procedure after obtaining only 1 good result in 5 cases. Phrenicoexeresis [57b'] has also been done, but the results were unsatisfactory. In the 21 collected

---

de Heyrovsky), Bol. Soc. de med. e cir. de São Paulo **15**:229, 1931. (*o*) von Haberer, H.: Beitrag zur kardiotonischen Speiseröhrenerweiterung, Zentralbl. f. Chir. **58**: 2947, 1931. (*p*) Urrutia, L.: Cuestiones gastroenterológicas, San Sebastian, Spain, Nueva Editorial, 1931. (*q*) Fromme, A.: Ueber weitere Erfahrungen bei der Behandlung des Kardiospasmus, Zentralbl. f. Chir. **60**:3632, 1933. (*r*) Hansen, J.: Beitrag zum Kardiospasmus des Kindesalters, Beitr. z. klin. Chir. **157**:617, 1933. (*s*) Churchill, E. D.: Oesophageal Surgery, Surg., Gynec. & Obst. **60**:417, 1935. (*t*) Haglund, A. L.: Esophago-Gastrostomies According to Method of Heyrovsky, Acta chir. Scandinav. **76**:109, 1935. (*u*) Ferrari, R. C., and Itoiz, O. A.: La esofagogastrostomía u operatión de Heyrovsky, Bol. y trab., Soc. de cir. de Buenos Aires **20**:773, 1936. (*v*) Brea, C. A.: Megaesófago por cardioespasmo. Su tratamiento por la esophagogastrostomía, ibid. **21**:733, 1937. (*w*) Calcagno, B. N., and Belleville, G.: Megaesöfago. Esofagogastrostomía, ibid. **21**:747, 1937. (*x*) Womack, N. A.: Esophagoplasty for Esophageal Achalasia, S. Clin. North America **18**:1241, 1938. (*y*) Notti, P., and Miyara, S.: Operación de Heyrovsky. Su applicación y resultado in un caso de estenosis del esófago abdominal (cardioespasmo), Bol. y trab., Soc. de cir. de Buenos Aires **23**:298, 1939. (*z*) Harris, R. I., cited by Janes.[81] (*a'*) Wilson, G., cited by Janes.[81] Bull.[1b] Janes.[81] Heyrovsky.[16b] Footnotes 57 *o*, *w* and *l'*. Footnotes 67 *m*, *c'*, *d'*, *w'*, *v'*, *w'* and *j''*. Frey.[80] Gröndahl.[81] Lambert.[82] Keller.[83] Footnote 84. Fromme.[87]

92. Braine and Metivet, cited by Grégoire.[71d]

93. von Hacker, cited by von Hacker and Lotheissen.[1d]

94. Footnotes 67*e'* and *f'*. Sencert and Simon.[71e]

cases [95] in which phrenotomy and transposition of the esophagus were done there were no deaths, and the results were stated as good in 12 (57.1 per cent), as showing improvement in 3 (14.3 per cent), and as failures in 6 (28.5 per cent). It is significant that in 2 of the cases in which improvement took place the authors regretted that they had not performed a plastic procedure.[96] Also of interest is the fact that among the cases in which the results were good there was only 1 with a follow-up longer than a few months.

As has been stated, a number of clinical and experimental observations have been presented in an attempt to explain the pathogenesis of achalasia on the basis of a nervous mechanism. It was therefore assumed that the condition might be corrected by surgical procedures directed at the nerve supply of the esophagus. In general, such procedures may be classified into those attacking the vagus nerves and those attacking the sympathetic nervous system. In 1911, Meyer [97] performed esophageal plication but in addition emphasized the importance of division of the vagal fibers entering the esophagus. However, the results in his cases were poor, and he finally concluded that the best treatment is cardioplasty. Sauerbruch [84a,b] also performed bilateral vagotomy through a transpleural approach in 4 cases. One of the patients died of pleuritis; 1 had a recurrence after a year, and the other 2 had recurrences after a very short period. On the basis of his experience Sauerbruch concluded that vagotomy is not justified and that its rationale is based on a false conception. Rieder's [98] patient became worse after the operation. Pieri,[99] in 1932, through a posterior mediastinotomy iso-

95. (*a*) Lardenvois, G., and Braine, J.: Phrénospasme chronique lié à un ulcus gastrique juxta-cardiaque. Dysphagie chronique grave avec dilatation de l'œsophage. Echece de dilatations répétées. Gastrostomie. Persistance des troubles. Découverte opératoire par voie thoracique de la traversée phrénique de l'œsophage. Débridement de l'orifice diaphragmatique. Guérison de la dysphagie, Bull. et mém. Soc. nat. de chir. **49**:937, 1923. (*b*) Kummer, E.: Sténose du segment diaphragmatique de l'œsophage. Phrénotomie par le procédé de M. Raymond Grégoire (thoracolaparotomie extraséreuse) Guérison, ibid. **53**:231, 1927. (*c*) Léfèvre and Jonchères: Cardiospasme de l'œsophage traité par la voie thoracoabdominale extra-séreuse de Grégoire, Bull. et mém. Soc. de méd. et de chir. de Bordeaux (1926), 1927, pp. 263-267. (*d*) Hartmann, in discussion on Delbet.[67x] (*e*) Chenut, M. A.: Un case de mégaœsophage traité par l'opération de R. Grégoire, Bordeaux chir. **3**:510, 1932. (*f*) Rochet, P.: A propos de l'opération de Heller pour cardiospasme, Lyon chir. **36**:389, 1939. Scrimger.[8e] Enderlen.[57b'] Footnotes 67 *l*, *s*, *t* and *x'*. Braine.[75d] Prat.[86a] Braine and Metivet.[92]

96. Prat.[86a] Léfèvre and Jonchéres.[95c]

97. Footnotes 40 and 41.

98. Rieder, W., discussion on Fromme,[67w] Arch. f. klin. Chir. **157**:137, 1929; Klinik und Therapie des sogenannten Cardiospasmus, Deutsche Ztschr. f. Chir. **222**: 47, 1930; footnote 23 *c*.

99. Pieri, G.: Sulla cura del cardiospasmo, Cong. Soc. internat. de chir., Rap. **1**:778, 1932; Contributi clinici alla chirurgia del sistema nervoso vegetativo; la cura della acalasia esofagea (cardiospasmo), Arch. ital. di chir. **35**:644, 1933.

lated both vagus nerves and sectioned the fibers entering the lower part of the esophagus. One of the patients, who also had a carcinoma, died of starvation and cachexia several months after operation. A satisfactory result was obtained in a second case, although the patient had empyema. That Pieri was not entirely convinced of the value of this procedure is evinced by the fact that he performed the Heller procedure in 2 subsequent cases. Jirasek [57b] reported a case in which the immediate result following bilateral vagotomy was good but in which recurrence subsequently took place. In the 11 collected cases in which vagotomy was done, 3 patients died; there were 7 recurrences, and a satisfactory result was noted in only 1 instance.

On the assumption that the obstruction is due to a functional disturbance of Auerbach's plexus, which innervates the circular musculature of the cardia, Recalde,[100] in 1924, advocated decortication of the plexus. In this way he attempted to interrupt the sympathetic nerve supply of the cardia. Of the 4 patients on whom he performed this operation, 1 died of peritonitis following accidental perforation of the esophageal musculature, and the other 3 had good results. One of these had been operated on sixteen years previously by the Heller technic and had had a recurrence. A number of observers had indicated that the sympathetic nervous system may play a significant role in the pathogenesis of this condition. However, it was not until 1934 that this contention was placed on a firmer support by the results of Knight's [101] experimental investigations. He showed that in cats there exists a true sphincter mechanism at the cardia; that bilateral vagal section reproduces the appearance of achalasia, which can be prevented by simultaneous removal of the sympathetic fibers, and that the condition can be relieved by sympathectomy after it has been produced experimentally. On this basis sympathectomy was performed in 5 cases of achalasia reported by Knight [102] and Adamson.[103] Because in dissections of cadavers it was found that the sympathetic nerve supply to the cardiac sphincter is derived chiefly from the left side of the celiac plexus and distributed along the left gastric artery, Knight advocated excision

100. Recalde, J. F.: (*a*) Sobre um caso de mal de engasgo curado cirurgicamente, Bol. Soc. de med. e cir. de São Paulo **7**:78, 1924; (*b*) Cardiospasmo, disfagia e megaesofago; simpaticectomia esofagica (statistica chirurgica), Arch. ital. di chir. **32**:613, 1932.

101. Knight, G. C.: The Innervation of the Esophagus, Proc. Roy. Soc. Med. **28**:891, 1935; footnote 22 *c*.

102. Knight, G. C.: Sympathectomy in the Treatment of Achalasia of the Cardia, Brit. J. Surg. **22**:864, 1935; Sympathectomy for Achalasia of the Cardia, Proc. Roy. Soc. Med. **28**:897, 1935.

103. Adamson, W. A. D.: Sympathectomy for Achalasia of the Cardia, Proc. Roy. Soc. Med. **28**:892, 1935.

of this artery, with its surrounding fat and nerve tissue. Of the 5 patients reported on by Knight and Adamson and treated in this manner, 1 was completely relieved, 1 was considerably improved and the other 3 showed signs of recurrence. Whereas the majority of surgeons have employed the procedure of Knight in the attack on the sympathetic nervous system, Craig, Moersch and Vinson [104] used a different technic. They performed sympathectomy by bilateral resection of the cervicothoracic sympathetic ganglions and trunk, with a good immediate result. Prior to the operation a diagnostic procaine hydrochloride block of the cervicothoracic sympathetic ganglions was done, and the patient was temporarily relieved, which indicated that the operation would be successful. In the 19 collected cases [105] of achalasia, including ours, in which sympathectomy was done there were 1 death [100b] due to peritonitis following accidental rupture of the mucosa and 1 due to suicide.[57l'] Recurrence took place in 4 cases, and improvement was only partial in 4. The results in these cases were not impressive. Mitchell [106] expressed the opinion that the high incidence of failures following these procedures may be explained on the basis of the great anatomic variation in the sympathetic nerve supply to the cardia and the consequent difficulty of interrupting the sympathetic pathways. In 1 of our cases a diagnostic procaine hydrochloride block by the anterior approach [107] was followed by rapid passage of barium sulfate through the cardia as determined fluoroscopically. Subsequently a bilateral cervicodorsal sympathectomy was performed, and, although the patient showed immediate improvement following the operation, several weeks later there was complete recurrence of the condition. The patient is to return for esophagogastrostomy.

Because of its anatomic location, adequate exposure of the cardia meets with relative technical difficulty. For this reason a number of surgical approaches have been devised. In general, these may be clas-

104. Craig, W.; Moersch, H. J., and Vinson, P. P.: Treatment of Intractable Cardiospasm by Bilateral Cervicothoracic Sympathetic Ganglionectomy: Report of a Case, Proc. Staff Meet., Mayo Clin. **9**:749, 1934.

105. Rupp, in discussion on Frey,[80b] Arch. f. klin. Chir. **186**:19, 1936. Souttar, H. S.: Oesophageal Obstruction, Brit. M. J. **2**:777, 1935. Eliason, E. L., and Erb, W. H.: Cardiospasm: Report of Two Cases Treated by Resection of Sympathetic Supply to the Cardiac Sphincter, Am. J. Surg. **35**:105, 1937. Stubbe, H.: Oesophageal Achalasia Treated by Sympathectomy, M. J. Australia **2**:1001, 1937. Meade, H. S.: A Case of Sympathectomy in the Treatment of Achalasia of the Cardia, Irish J. M. Sc., 1939, p. 130. Gray and Skinner.[57l'] Recalde.[100] Knight.[101] Knight.[102] Adamson.[103] Craig and others.[104]

106. Mitchell, G. A. G.: The Nerve Supply of the Gastro-Oesophageal Junction, Brit. J. Surg. **26**:333, 1938.

107. Ochsner, A., and DeBakey, M.: Treatment of Thrombophlebitis by Novocaine Block of Sympathetics, Surgery **5**:491, 1939.

sified into transabdominal and transthoracic.[108] Of the former there are two types: those in which exposure is obtained through a high median or a left paramedian incision and those in which the left costal arch is mobilized. The majority of surgeons have used the former, and in our opinion this is usually sufficient. In some cases this is combined with partial mobilization of the costal cartilage near the sternum and division of this cartilage at this site.[109] Others have preferred a subcostal incision parallel to the costal margin, similar to that described by Fenger in 1854. Still others have contended that a more satisfactory exposure of the cardia may be obtained by mobilization of the costal arch. In 1887, Lannelongue [110] proposed mobilization of the costal arch by resection of segments of the lower four or five ribs and their cartilages anteriorly. Working on cadavers, Micheli,[111] in 1895, modified this procedure by using a Schede type of incision and simply dividing the ribs medially and laterally so that the resultant flap could be thrown up. In 1898, von Mikulicz [112] employed a somewhat similar approach in a case of carcinoma of the cardia, and in another case he used multiple transverse incisions over the respective costal cartilages in order to expose and divide them. Somewhat similar approaches were used more recently by Lambert [82] and by Churchill.[91s] Such procedures are so extensive that the approach alone is sufficient to put the patient in shock. In fact, Wiener,[113] in 1908, suggested dividing the operation into stages, with mobilization of the costal arch as the first stage. Kelling also desired to avoid the shock associated with these procedures but proposed a method which was apparently worse. In 1901 he [114] recommended increasing the subdiaphragmatic exposure by the patient's posture, recommending one in which the lower part of the abdomen and the extremities hung vertically over the end of the table. Apparently

108. Ochsner, A., and DeBakey, M.: Carcinoma of the Esophagus, J. Thoracic Surg., to be published.

109. Ferrari, R. C.: La esófago-gastrostomía por vía abdominal u operación de Heyrovsky. Técnica operatoria, Semana méd. **1**:1539, 1936; La cardiotomía extramucosa (operación). Técnica operatoria, ibid. **1**:1081, 1936. Clute, H. M., and Albright, H. L.: Cutting the Costal Arch for Upper Abdominal Exposure, Surg., Gynec. & Obst. **67**:804, 1938. Footnotes 91 *m*, *v* and *w*.

110. Lannelongue: Les abscès tuberculeux périhépatiques et leur traitement, Semaine méd. **7**:235, 1887.

111. Micheli, cited by Wiener.[113]

112. von Mikulicz, J.: Beiträge zur Technik der Operation des Magencarcinoms, Verhandl. d. deutsch. Gesellsch. f. Chir. **2**:252, 1898; Beiträge zur Technik der Operation des Magencarcinoms, Arch. f. klin. Chir. **57**:524, 1898.

113. Wiener, J.: Osteoplastic Resection of the Costal Arch Followed by Resection of Lesser Curvature of Stomach and Esophagus and Esophagostomy, Ann. Surg. **48**:530, 1908.

114. Kelling, G.: Becken-Hängelage bei horizontalem Rumf für Operationen in der Nähe des Zwerchfells, Zentralbl. f. Chir. **28**:1025, 1901.

even he found this unsatisfactory, because several years later he suggested another posture.[115] Although Marwedel's [70] and Asthoewer's [116] publications appeared in the same year, 1903, the former preceding the latter by a few months, Asthoewer apparently deserves priority. He stated that in 1894 he operated on a patient with fibroma of the spleen. He first made a left rectus incision, but because of the size of the tumor it was necessary to obtain greater exposure. He accomplished this by converting the incision into the shape of a U and then dividing the ribs from the eighth to the tenth laterally and their fused cartilages medially. The resultant osteoplastic flap could be mobilized upward. In a second case, in which there was a sarcoma of the left side of the thoracic wall, he used a paracostal incision and divided the seventh costal cartilage near its attachment to the sternum and the eighth through the tenth ribs in the midaxillary line. Marwedel used a paracostal incision and through this exposed and divided the seventh cartilage near the sternum and the seventh, eighth and ninth ribs near the chondrocostal junction. In 1906, Meyer [117] employed a similar approach but made a right-angled incision. Undoubtedly mobilization of the costal arch increases the exposure, but because of the increased extent and duration of operation its use is seldom justified. Moreover, because of mobilization of the chest wall there is greater likelihood of postoperative morbidity.

Transpleural Approach.—Although Biondi,[118] in 1895, suggested the transpleural approach for resection of the cardia for malignant disease of this portion of the esophagus, Gosset,[79] in 1903, advocated the transpleural approach for resection of the cardia for nonmalignant disease of this area. He performed this operation on cadavers and on animals. The operation which he proposed and performed experimentally consisted of a side to side anastomosis between the dilated esophagus and the fundus of the stomach. Meyer,[97] in 1911, performed vagolysis of the esophagus transpleurally. Because of a recurrence the following year, he performed a transpleural cardioplasty. Henschen,[119] working in Sauerbruch's clinic, performed the first successful esophagogastrostomy by the transpleural route. This case was subsequently

115. Kelling, G.: Technische Beiträge zur Chirurgie der Bauchhöhle, Zentralbl. f. Chir. **31**:90, 1904.

116. Asthoewer: Die Aufklappung des Rippenbogens zur Erleichterung, operativer Eingriffe im Hypochondrium und Zwerchfellkuppelraum, Zentralbl. f. Chir. **30**:1257, 1903.

117. Meyer, W.: Osteoplastic Resection of the Costal Arch in Order to Reach the Vault of the Diaphragm, J. A. M. A. **47**:1069 (Oct. 6) 1906. Meyer, H. W.: Osteoplastic Resection of the Costal Arch for Gunshot Wound of Spleen, Surg., Gynec. & Obst. **48**:412, 1929.

118. Biondi, D.: Esofago-gastrostomia sperimentale intrathoracia, Policlinico (supp.) **1**:964, 1895.

119. Henschen, in discussion on Frey,[80b] Arch. f. klin. Chir. **186**:20, 1936.

referred to in Sauerbruch's publication. Denk,[84g] in 1918, reported an esophagogastrostomy performed by the transpleural route. Sauerbruch popularized the transpleural approach for operations on the cardiac end of the esophagus. The advantages of the transpleural approach are obvious, particularly since it facilitates exposure of the cardiac end of the esophagus and the upper portion of the stomach. This is due to the upward arching of the diaphragm, which makes exposure of this portion of the digestive tract from the abdomen relatively difficult. Another advantage of the transpleural approach is that it is possible to cut or crush the phrenic nerve at the time of the operation without any difficulty. On the other hand, transpleural exposure of the lower end of the esophagus is fraught with many dangers and is more complicated than the usual transabdominal approach. The danger of infection of the pleural cavity, particularly if the esophageal mucosa is open, is much greater than the danger of peritonitis. Of 16 patients [120] operated on by the transpleural approach, 5 died (31.2 per cent). An additional patient had a pleural infection.

A combination of transthoracic and transabdominal approach was suggested in 1933 by Ohsawa,[74] who designated it as a free transdiaphragmatic thoracolaparotomy. The operation was performed through a hook-shaped incision beginning at the junction of the anterior axillary line and the seventh rib, crossing the level of the tenth rib at the posterior axillary line and terminating at the level of the sixth rib at the paravertebral line. The seventh and eighth ribs were resected over a distance of about 7 cm., and the thoracic cavity was entered. After the esophagus had been mobilized, the abdomen was opened by incising the diaphragm from the hiatus outward. This approach undoubtedly gave an excellent exposure, but it appears to us that because of the magnitude of the procedure its use is hardly justified for benign lesions. Grégoire,[121] in 1923, proposed an extraserous approach exposing both the thoracic and the abdominal cavity. There are the same objections to this type of procedure as to the transpleural and the complicated abdominal approaches.

Anesthesia.—The majority of patients with achalasia who have been operated on have had general anesthesia. Finsterer,[91e] Fromme [67w] and Fruchaud [67p'] advocated splanchnic analgesia combined with either local or light ether anesthesia. South American and Spanish surgeons have preferred spinal analgesia. Although one of our esophagogastrostomies was done with the patient under general anesthesia, we are of the opinion that spinal analgesia induced with properly administered nupercaine

120. Footnotes 67 *w*, *c'* and *d'*. Frey.[80] Footnotes 84 *a*, *b*, *d*, *e* and *f*. Prat.[86a] Fromme.[87] Meyer.[90] von Haberer.[91o]

121. Footnotes 71 *d*, *e*, *f*, *g*, *h*, *i* and *j*.

and carefully supervised by a trained anesthetist, as used in our case 2, is the anesthesia of choice. This permits adequate relaxation and analgesia of sufficient duration for the operation to be completed without difficulty.

REPORT OF CASES

CASE 1.—Mrs. A. D., a white woman 44 years of age, was admitted to the Hutchinson Memorial Clinic Sept. 11, 1934, complaining chiefly of difficulty in swallowing, which began suddenly approximately twenty-two years previously. At this time she was three months pregnant. She had difficulty in swallowing both liquids and solids, and the dysphagia was associated with retrosternal pain, dyspnea and smothering sensations. She soon learned that by drinking large quantities of water rapidly immediately after eating and standing erect and straining during expiration she could force some food down. The condition continued with exacerbations until 1932, when a gastrostomy became necessary for feeding purposes. She was treated by bougienage and was temporarily relieved, but the condition soon recurred. She had lost about 20 pounds (9 Kg.) in weight. The past history, family history and social history were not significant. Physical examination revealed her to be thin, poorly nourished and of hyposthenic habitus. She was intelligent and cooperative, with no evidence of nervousness or mental depression. The temperature, pulse rate and respiratory rate were normal. The blood pressure was 120 systolic and 80 diastolic. Detailed physical examination revealed no gross abnormalities except emaciation, retrocele and cystocele.

Special Tests.—Urinalysis revealed no abnormalities. The Wassermann reaction was negative. There were 4,000,000 red blood cells per cubic millimeter, with 80 per cent hemoglobin; the white blood cell count was 4,000 per cubic millimeter, with 68 per cent neutrophils.

Roentgen Data.—Fluoroscopic examination of the esophagus after ingestion of barium sulfate (September 14) revealed a marked dilatation of the entire esophagus, with an apparent obstruction at the cardia as only a small amount of barium trickled through into the stomach. The esophagoscope revealed the esophagus markedly dilated and filled with large quantities of foul material. There was no other organic change.

Progress.—The patient was treated by dilation with mercury bougies. This produced temporary improvement, and she was discharged. However, the condition recurred, and she was readmitted in February 1935. Bougienage therapy again resulted in temporary improvement, and she returned home. She was readmitted a year later with recurrence of the condition and was again treated with mercury bougienage, with results similar to those obtained on previous occasions.

She returned to the hospital in August 1937, stating that her condition was much worse. Roentgen studies on August 7 revealed marked dilatation and tortuosity of the esophagus, with obstruction at the cardia (fig. 11 *A*). On August 18 bilateral procaine hydrochloride block of the stellate ganglions was performed by the anterior approach. Immediately afterward fluoroscopic examination of the esophagus following ingestion of barium sulfate revealed free and rapid passage of the barium mixture through the cardia into the stomach. This indicated that the patient might be relieved by sympathectomy. On August 20, with general anesthesia, the left stellate and the second and third thoracic sympathetic ganglions and the intervening trunk were excised. About ten days later

a similar procedure was performed on the right side. The postoperative course was uneventful, and the patient was able to swallow much better when she was discharged, on October 5. On September 21 roentgen examination following ingestion of a barium sulfate meal had revealed the esophagus as much dilated as before operation, but there was some improvement in the passage of barium through the cardia (fig. 11 *B*).

The patient was again observed in February 1938, and at this time her condition was apparently no better than it had been prior to operation. She had

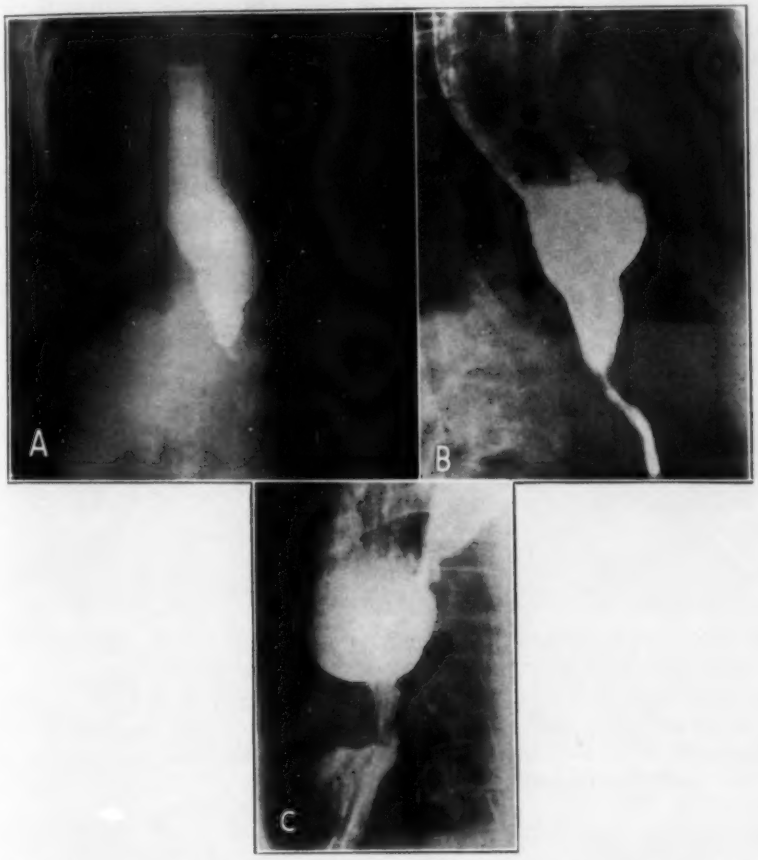


Fig. 11 (case 1).—*A*, roentgenogram of the esophagus following ingestion of a barium sulfate meal, showing marked dilatation and tortuosity, with obstruction at the cardia. *B*, roentgenogram of the esophagus and cardia after ingestion of a barium sulfate meal approximately one month after sympathectomy. The dilatation is still present, but there is some improvement in the passage of the barium through the cardia. *C*, roentgenogram of the esophagus and cardia after ingestion of a barium sulfate meal three years after sympathectomy. The dilatation and obstruction remain unchanged.

gained no weight, and there was complete recurrence of the dysphagia. She was again treated by mercury bougienage, with temporary improvement.

She returned to the hospital on July 16, 1940, and roentgen studies revealed the condition to be unchanged (fig. 11 *C*). She is to return for esophagogastrostomy.

CASE 2.—Mrs. E. S., a white woman 63 years of age, was admitted to Touro Infirmary on Aug. 9, 1939, complaining chiefly of fulness and vomiting after eating small amounts of food. This began thirty-three years previously, and progressively became worse until, at the time of admission, she had difficulty in swallowing fluids. She occasionally had attacks of severe retrosternal pain associated with marked dyspnea. Nine years previously she had been operated on and told that she had an inoperable carcinoma of the stomach. For the past year she had been treated by mercury bougienage with little improvement. She had lost a considerable amount of weight. The past history, social history and family history were not significant.

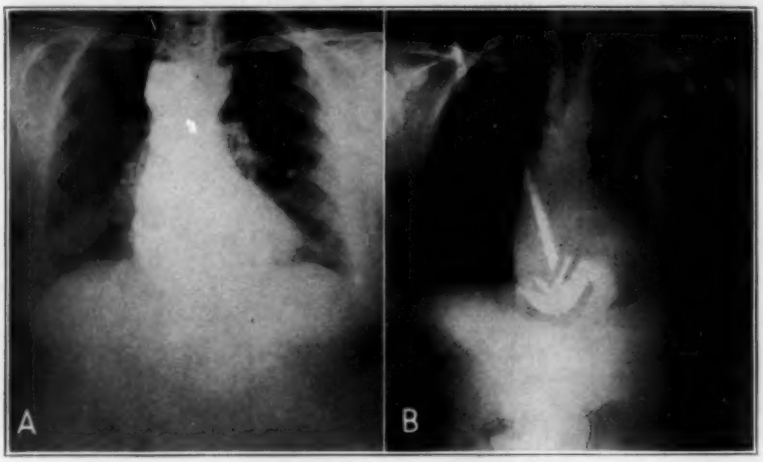


Fig. 12 (case 2).—*A*, roentgenogram of the esophagus after ingestion of a barium sulfate meal, showing enormous dilatation and marked tortuosity of the esophagus, with obstruction at the cardia and practically no leakage of barium into the stomach. *B*, roentgenogram of the esophagus following ingestion of barium sulfate meal after operation. There is a distinct decrease in dilatation, and the barium mixture passes freely and rapidly into the stomach.

Physical examination revealed her to be fairly well developed. She did not appear acutely ill. She was intelligent, cooperative and cheerful. The temperature, pulse rate and respiratory rate were normal. The blood pressure was 120 systolic and 80 diastolic. Detailed physical examination revealed no gross abnormalities.

Special Tests.—Urinalysis revealed a few pus cells. The Wassermann reaction was negative. The blood picture was normal. The renal function test revealed no abnormality. The electrocardiogram showed slight evidence of myocardial changes. Roentgen studies following ingestion of a barium sulfate meal revealed considerable dilatation and tortuosity of the esophagus. There was definite constriction of and obstruction to the passage of barium at the cardia (fig. 12 *A*).

Esophagoscopic study showed the esophagus to be markedly dilated and to contain a large quantity of foul material. There were no other abnormalities, and the cardia could be entered readily.

Operation.—After adequate preparation, on September 5, with nupercaine spinal analgesia through a left subcostal incision an esophagogastrostomy as described and illustrated in figures 8 to 10 was performed.

Progress.—The postoperative course was uneventful, and the patient was on a regular diet within ten days after operation. She was discharged on September 22, in excellent condition. She had no difficulty in swallowing any food.

The patient was observed on July 5, 1940, ten months after operation, and was found to be in excellent condition. She was able to eat everything with no difficulty and had been completely relieved of all previous manifestations. She had gained 35 pounds (16 Kg.). Roentgen studies following ingestion of barium

Fig. 13 (case 3).—*A*, roentgenogram of the esophagus after ingestion of a barium sulfate meal, showing marked dilatation of the esophagus and constriction at the cardia, with little seepage of barium into the stomach. *B*, roentgenogram of the esophagus after ingestion of barium sulfate after operation. There is definite diminution in the size of the esophagus, and the barium flows readily into the stomach.

sulfate revealed some dilatation, which was less than that observed prior to operation. There was rapid passage of the barium mixture through the cardia (fig. 12 *B*).

Case 3.—B. B., a white boy 9 years of age, was admitted to the Charity Hospital on Nov. 29, 1939, complaining chiefly of vomiting food recently ingested. He had begun to have symptoms about one year previously, when it was observed that after eating a part of his meal he had the sensation that it had stopped in the lower part of his chest and that if more were eaten regurgitation would occur. This was not associated with pain or nausea. The condition progressively became worse, with intermittent attacks during which he could swallow only milk. The past history and the family history were not significant.

Physical examination revealed him to be slightly undernourished for his age but quiet, cheerful and cooperative. There were no gross abnormalities. The temperature, pulse rate and respiratory rate were normal. The blood pressure was 105 systolic and 65 diastolic. Detailed physical examination revealed no gross abnormalities.

Special Tests.—Urinalysis showed no abnormalities. The Wassermann reaction was negative and the blood picture normal. Roentgen studies on July 8 after a barium sulfate meal revealed marked dilatation of the esophagus with obstruction at the cardia and slight seepage of barium into the stomach (fig. 13 *A*). The esophagoscope showed the esophagus markedly dilated and containing large quantities of foul material. There was no other organic change, and passage through the cardia was easily done.

Progress.—The patient was treated conservatively by mercury bougienage and antispasmodics for several months, with only slight temporary improvement. After this he was discharged but returned to the hospital on May 9 in a worse condition, and it was decided that an operation was necessary. Accordingly, after necessary praparation, on July 11, 1940, with general inhalation anesthesia, through a left subcostal incision an esophagogastrostomy was performed as described and illustrated in figures 8 to 10. The postoperative course was uneventful. One week after operation he was swallowing all liquids normally and within another week he was on a regular diet. He had no complaints and was discharged on July 26. Roentgenologic studies following ingestion of barium sulfate on July 23 had revealed a distinct decrease in the dilatation of the esophagus and free and rapid passage of the barium mixture into the stomach (fig. 13 *B*).

The patient was again observed on August 10 and was found to be in excellent condition, with complete relief of all previous manifestations.

SUMMARY

The various factors which have been considered of pathogenic significance in the development of achalasia of the esophagus are briefly reviewed.

The various types of radical procedures which have been advocated and employed are classified into four large groups, depending on whether they are directed at (1) the dilated esophagus; (2) the cardia; (3) the diaphragm or (4) the nerve supply. A brief historical consideration of each procedure is presented. The various operations are described and illustrated, and the collected cases are analyzed.

The procedures directed at the dilated esophagus and based on an attempt to reduce the size of circumference by esophagoplication are irrational and are considered of historical interest only.

Four types of procedures have been directed at the cardia: (1) dilation; (2) plastic operation; (3) excision, and (4) deviation.

Dilation of the cardia has been done by retrograde bougienage and transgastrically by instruments or fingers. In 80 cases collected from the literature in which the latter procedure was used 7 (8.9 per cent) patients died and 8 (10.1 per cent) operations were failures.

The plastic procedures consist of extramucous cardiomyotomy or cardioplasty. In 104 collected cases in which the former was employed there were 4 deaths and 14 recurrences. In 36 cases in which the latter was used there was 1 death and 1 recurrence.

Excision of the cardia followed by esophagogastrostomy has been done in 2 cases, in 1 of which the patient recovered. Such a radical procedure, in our opinion, is justified only in the presence of a malignant tumor.

Of the various procedures directed at the cardia, esophagogastrostomy is considered the most rational. This may be performed either by side to side anastomosis between the esophagus and the fundus of the stomach or, preferably, by an anastomosis, similar to the Finney gastroduodenostomy, which obviates the cardiac spur in the esophagus and thus creates a wider opening between the esophagus and the stomach. In 88 cases collected from the literature in which esophagogastrostomy was performed there were 5 deaths (6.6 per cent) and only 1 poor result.

Operations directed at the diaphragm consist of phrenotomy and mobilization of the esophagus downward. In 21 collected cases in which these measures were used there were no deaths, and the results were stated as good in 12 (57.1 per cent), showing improvement in 3 (14.3 per cent) and failures in 6 (28.5 per cent).

The procedures directed at the nerve supply may be classified into those attacking the vagus nerves and those attacking the sympathetic nervous system. In 11 collected cases in which operations of the former type were done, 3 patients died, 7 recurrences were observed and only 1 satisfactory result was recorded. In 19 collected cases in which operations of the latter type were done there were 1 death due to peritonitis and 1 to suicide. There was recurrence in 4 cases, and partial improvement was observed in 4.

Three cases are reported by us. An esophagogastrostomy was done in 2, with excellent results. A sympathectomy was performed in the other, with recurrence.

SURGICAL TREATMENT OF CARCINOMA OF THE ESOPHAGUS

JOHN H. GARLOCK, M.D.
NEW YORK

The importance of carcinoma of the esophagus becomes evident when it is realized that in New York city alone it accounts for 3.5 per cent of all deaths from cancer (Watson). In the United States registration area the incidence of cancer of the esophagus increased almost 100 per cent from 1915 to 1932. Clairmont, in 1924, estimated that about 25,000 persons die of this disease annually in Europe. With the realization that cancer of the esophagus is assuming increasing importance from the standpoint of numerical incidence and with the rapid strides being made in the fields of anesthesia and thoracic surgery has come an ever increasing interest in the radical surgical treatment of this disease, based on the present day conception of surgical treatment of cancer in general. It is, however, only in recent years that encouraging progress has been noted. Although at present operative treatment of cancer of the esophagus is being carried out by a relatively small group of surgeons, it is my prediction that within a very few years such treatment will be the accepted method of handling this disease and will be universally adopted.

It must be remembered that, from the pathologic standpoint, carcinoma of the esophagus is usually a slowly growing tumor and that it remains a local disease for some time before metastasizing to the regional lymph nodes. However, although it may not spread peripherally until late, it may become locally inoperable in the early stages because of fixation to a nearby vital structure, such as the aorta or a bronchus. The importance of diagnosis early in the disease needs little discussion. More rapid advance in the surgical treatment of cancer of the esophagus will be made when increasing numbers of early lesions are referred to the surgeon. It is important to stress, therefore, that the physician must regard with suspicion any disturbance in the act of swallowing in a patient past 35 or 40 years of age. Persistent dysphagia calls for careful roentgen examination. In cases of early involvement the roentgenogram may reveal nothing abnormal, especially if the ordinary barium sulfate mixture is used. If a heavier mixture of barium is employed, the mucosal pattern of the esophagus will be more accurately depicted, and an early lesion will be more easily demonstrated. Roentgen examination should include fluoroscopic observations of the activity of the

From the Surgical Service of the Mount Sinai Hospital.

esophagus during the act of swallowing the mixture of barium. Fixation of a portion of the esophageal wall, such as would occur in the presence of a tumor, would be an important diagnostic point. This examination should be supplemented by esophagoscopy. The purpose of this examination is to determine the presence of a neoplasm, to note its gross appearance, to obtain a biopsy specimen of the tumor and to localize the position of the growth with reference to the distance from the upper incisor teeth. The last-mentioned point is of importance because it helps the surgeon to decide which type of operation should be undertaken. No patient should be subjected to radical operation unless positive results have been obtained for biopsy.

Until recent years the diagnosis of cancer of the esophagus was frequently made late in the disease, too late for surgical treatment, and such a diagnosis was synonymous with a fatal prognosis. The accepted method of treatment was by radiation (roentgen and radium), supplemented usually by gastrostomy. An enormous experience with these modalities has accumulated throughout the world, and critical survey of the results is possible. It is fair to state that the results of radiation therapy for cancer of the esophagus have been universally disappointing. Of the vast number of patients treated by these methods, approximately 20 are said to be cured, and the cure of many of these is open to question, either because the follow-up period has been too short or because histologic evidence of the presence of carcinoma has been incomplete or inconclusive. That radiation therapy, whether by local use of radium or by external irradiation, is not without considerable danger is indicated by the numerous reports of perforation of the esophagus with fatal mediastinitis. In a fairly large experience with this disease, I have never seen a case in which radiation therapy has prolonged life beyond the period of the usual life history of the disease, provided that a gastrostomy has been performed for feeding purposes. Ricard and Ballivet crystallized the general opinion among surgeons today when they said that if surgeons had a chance to resect the large number of early lesions now being referred to radiologists, their results would be incomparably better than any the radiologists have been able to report.

Unfortunately, many patients, with cancer of the esophagus are in the sixth and seventh decades of life and associated degenerative diseases of the heart, kidneys and blood vessels may preclude any attempt at radical surgical therapy. It would be foolhardy to subject a patient in his seventies, with a poor heart, arteriosclerosis and poorly functioning kidneys, to the hazards of so extensive a procedure. On the other hand, many patients in these age groups are in excellent condition and can be brought safely through the surgical ordeal. The patient in Torek's famous case is a noteworthy example. Each case should be judged on

its own merits, and the surgeon should have the advice and counsel of a competent physician to aid him in estimating the physical equipment of the patient. The surgical treatment of esophageal cancer is a cooperative problem, involving not only the surgeon but the physician, the esophagoscopist, the anesthetist, the nursing staff and the operating room personnel.

PREOPERATIVE PREPARATION

It is most important to prepare the patient thoroughly, so that the risk of the operative procedure may be minimized. Careful attention to oral hygiene will lessen the possibility of pulmonary complications incident to prolonged anesthesia and manipulation of the lobes of the lung. Carious teeth should be extracted and the gums and teeth cleansed. A high caloric liquid diet containing the necessary vitamins and minerals should be given. To combat dehydration, which is frequently present, fluids should be given by venoclysis as well as by mouth. A daily intake of 3,000 cc. is desirable. A transfusion of 500 cc. of whole blood should be given a day or two prior to operation. As the surface of the carcinoma is ulcerated and covered with highly infectious organisms, every effort should be made to decrease the virulence and number of these bacteria so as to minimize the possibility of mediastinal or pleural infection. I have approached this problem in two ways. As a result of a study of the value of preoperative administration of sulfanilamide in surgical treatment of the colon (with bacteriologic controls), I have routinely administered sulfanilamide to patients with cancer of the esophagus for seventy-two hours prior to operation, in 15 grain (0.97 Gm.) doses every four hours. A large experience with these lesions will be the deciding factor in determining the efficacy of this drug as a prophylactic medicament. I cannot agree with the findings of Bricker and Graham that preoperative administration of this drug interferes with wound healing. The only objection to its use that has been encountered, and it is a minor one, is that the cyanosis produced by the drug may upset the anesthetist's estimation of the amount of oxygen required in administration of the anesthesia.

The second line of attack against the bacterial flora of the ulcerated neoplasm consists of mechanical cleansing of the esophagus by frequent irrigation. A Levine tube is passed to a point just above the neoplasm, and through it warm saline or boric acid solution is flushed past the growth. If a gastrostomy is present, the washings will emerge from the stomach. If no gastrostomy has been performed, the washings will either pass into the stomach or may be siphoned back. If these irrigations are repeated daily for four or five days prior to operation, a material change in the appearance of the ulcerating surface of the neoplasm will be noted.

A few authors have stressed the importance of pneumothorax before the operation in order to accustom the lung to the positive intrapleural pressure which takes place at the time of operation. I have never seen the need for this and have relied on my anesthetist to maintain varying degrees of positive intrapulmonary pressure. However, I can see no objection to preoperative pneumothorax provided that operation is not delayed too long. I do not subscribe to the recommendations of a few writers that the phrenic nerve be crushed either prior to or during the operation. Paralysis of the diaphragm adds another burden to an already handicapped patient in his effort to obtain maximum pulmonary ventilation.

GASTROSTOMY

If the neoplasm is located in the upper two thirds of the esophagus, a preliminary gastrostomy will be indicated.[1] Ten to fourteen days should elapse before resection of the organ is carried out. During this time, high caloric feedings containing all vitamins and minerals should be given through the gastrostomy opening. Marked improvement in the patient's general condition may be noted during this preparatory period, and there may be an appreciable gain in weight. The type of gastrostomy performed should be one that insures permanent patency at the cutaneous surface and one that is large enough to admit a no. 28 or a no. 30 French catheter. The Janeway gastrostomy meets these requirements but has the serious objection of frequent regurgitation of gastric contents through the opening. The valve gastrostomy described recently by Spivack apparently overcomes this deficiency. This procedure at present is the operation of choice.

If the carcinoma is located in the distal third of the organ, preliminary gastrostomy should not be carried out, because the presence of a gastrostomy will seriously interfere with the performance of an intrathoracic

1. Recently I have changed the plan of procedure just described. This was done in order to obviate regurgitation of gastric contents through the gastrostomy opening, which occurs even with the Spivack valvular gastrostomy. If the patient is in fairly good condition, a preliminary gastrostomy is not performed. Instead, during the thoracic part of the operation, the lower portion of the esophagus is divided about 2 inches (5 cm.) above the cardia, and this lower 2 inch segment, still attached to the stomach, is dissected from the mediastinum and pushed into the abdomen. (It is covered with a rubber envelope to prevent contamination.) After completion of the thoracic procedure, the abdomen is opened through a small left rectus incision and the esophageal stump is brought out at the upper angle of the wound and sewn to the skin to form an esophagostomy opening. In this way, the sphincter mechanism of the cardia is preserved and no leakage of gastric contents can take place. Another advantage of this new procedure is that the subsequent antethoracic esophagoplasty may be completed in one stage without fear of regurgitation of acid gastric secretion, which ordinarily causes digestion of the skin-lined tube.

anastomosis between the stomach and the esophagus, the operation of choice for neoplasms in this situation. For the same reason it is questionable whether a jejunostomy should be performed for feeding purposes. However, an occasional patient may require such a procedure in order to improve the general condition. If it becomes necessary, an incision at the level of the umbilicus or below it should be used, in order to remove the field of this operation as far as possible from the upper part of the abdomen.

ANESTHESIA

In my experience, the combination of avertin with amylene hydrate and ethylene or cyclopropane anesthesia has been most satisfactory. Ethylene is the least irritating of the inhalation anesthetics and causes little or no circulatory depression. The only objection to its use is that the actual cautery cannot be used to divide the esophagus during the operation. However, its many advantages far outweigh this minor inconvenience. The anesthetic agent must be administered with varying degrees of positive pressure during the operation to influence the extent of inflation of the lung. Complete collapse of the lung should not be permitted to take place at any time during the operative procedure. During the course of the operation, perhaps every ten minutes, it is advisable to have the anesthetist expand the lung by increasing the pressure of the anesthetic gas. Any circulatory or respiratory depression that may have occurred is quickly overcome. It is important that the services of a competent anesthetist be obtained. The incidence of postoperative pulmonary complications depends in large measure on the skill of administration of the anesthetic agent.

A number of authors recommend intratracheal anesthesia, stating that the degree of inflation of the lungs can be more accurately controlled by this method. I have felt that the trauma of intubating the trachea in these frequently debilitated patients may well be a factor in the development of pulmonary complications and have therefore had no experience with this method for esophagectomy. The results with ordinary positive pressure anesthesia have been satisfactory.

It has seemed convenient, in discussing the operative treatment of carcinoma of the esophagus, to divide the organ into thirds. The upper third, extending from the hypopharynx to the arch of the aorta, presents an altogether different surgical problem from that encountered in the middle and lower thirds. The middle third of the organ extends from the arch of the aorta to a point about 34 to 36 cm. from the upper incisor teeth. The length of this portion of the esophagus varies somewhat with the height of the patient. The lower third extends to the cardia. It becomes apparent that it is important for the esophagoscopist to locate accurately the position of the neoplasm with respect to the distance from

the upper incisor teeth. This information helps the surgeon to decide which operative procedure he will choose.

CARCINOMA OF THE UPPER THIRD OF THE ESOPHAGUS

Surgical treatment of cancer of the hypopharyngeal, cervical and supra-aortic portions of the esophagus has been a difficult problem, owing mainly to the fact that tumors in these situations are frequently overlooked and reach the surgeon when growth has taken place beyond the wall of the organ. When the tumor is located in the cervical portion of the organ, the operative procedure employed will depend on the extent of involvement of the esophagus itself and also of neighboring structures, such as the larynx. Small tumors may be removed through lateral esophagotomy, with preservation of part of the circumference of the esophageal wall. Healing takes place by granulation with subsequent stricture, which will require dilations. If the larynx is involved also, laryngectomy in addition to cervical esophagectomy is indicated. This necessitates a permanent tracheotomy in the suprasternal notch and subsequent plastic reconstruction of the esophagus by the use of sliding cervical skin flaps (method of Eggers). In most of the reported cases the regional lymph nodes have been excised, but in most instances they were found uninvolved.

When the supra-aortic portion of the esophagus is involved by cancer, fixation to the trachea or to the main bronchus of the left lung may take place early in the course of the disease. For this reason, King advised that bronchoscopy be performed preoperatively in order to exclude such invasion. Infiltration of the left recurrent nerve is a not infrequent finding. Successful resections for cancer in this situation have been few. Küttner freed a growth at the level of the clavicles and invaginated the lower stump, packing off the upper mediastinum. Zaaijer exposed this region by removing the manubrium, portions of the clavicle and the first two ribs on the left side, while Sauerbruch described a right anterior upper thoracic approach. The region under discussion may also be effectively exposed by an upper posterior mediastinotomy.

CARCINOMA OF THE MIDDLE THIRD OF THE ESOPHAGUS

Although a number of operations have been reported for malignant growths involving this portion of the esophagus, the procedure described by Torek has stood the test of time and experience. I have modified it in some respects in order to simplify the steps and shorten the operative time. The operation is based on the sound surgical principles of adequate exposure, free vision of the operative field and radical excision of the cancer-bearing focus with the associated lymph nodes.

As a result of some experimental and clinical studies, it has been determined that it is impossible to resect a portion of the thoracic part of the esophagus, perform an end to end anastomosis and obtain satisfactory union and continuity of the viscus. The reasons for this are: 1. In order to effect a radical removal, too large a section of the organ would necessarily be removed to permit of bridging the gap without great tension at the subsequent suture line. 2. The esophagus is devoid of peritoneal covering, which is important in satisfactory healing. 3. The longitudinal muscle fibers surrounding the esophagus exert so strong a pull that any type of anastomosis is quickly torn apart.

The operation is tedious and difficult. The steps of the procedure should be carried out methodically, without haste. It is important to avoid unnecessary bleeding and to prevent infiltration of the mediastinum with blood. This requires careful hemostasis. The lobes of the lung should be handled as gently as possible, and care should be exercised not to traumatize the arch and the descending portion of the aorta. These precautions will have the effect of diminishing operative shock.

Operative Technic.—The patient is placed on his right side with the head resting on the abducted right arm and his back close to the edge of the operating table. The left arm is elevated. One or two sandbags placed against the anterior region of the chest will prevent the patient from rolling forward on the operating table. After the usual preparation of the skin, the patient is draped in such a way as to leave exposed the chest, the upper part of the abdomen and the left shoulder and arm.

The incision begins in the seventh interspace in the midclavicular line and is continued posteriorly along the course of the seventh interspace (fig. 1). It is then carried upward between the vertebral border of the scapula and the spine as far as the third rib. The thoracic musculature is divided in the line of the cutaneous incision. All bleeding points are carefully ligated before the thoracic cavity is entered. An incision is made in the seventh interspace, dividing the parietal pleura with the intercostal muscles. The seventh, sixth, fifth and fourth ribs are quickly divided near the vertebral transverse processes, and the intercostal vessels are ligated with hemostatic sutures. A rib spreader is inserted, affording a clear view of the left thoracic cavity. The lobes of the lung usually collapse when the chest is opened, but complete collapse should not be permitted to take place. The arch and the descending aorta stand out in sharp relief. The esophagus lies to the right of the aorta, in the mediastinum.

A vertical incision is made in the mediastinal pleura from the under surface of the aortic arch down to the diaphragm (fig. 2). The pleural edges are dissected away from the underlying esophagus. It is not difficult to free the esophagus bluntly throughout its circumference at one point. A tape is placed around the organ at this site, and traction

permits the surgeon to free the esophagus throughout its extent (fig. 3.). It is important to ligate the few small esophageal arteries rising from the aorta and to divide the small branches of the vagus nerve. If

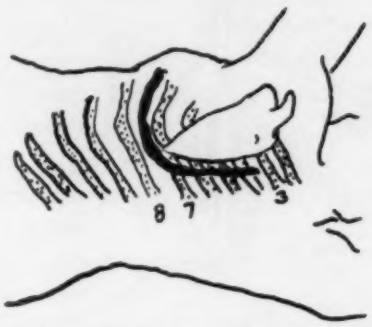


Fig. 1.—Incision is made in the seventh interspace, starting in the midclavicular line and carried posteriorly. It extends upward between the vertebral border of the scapula and the spinal column. The seventh, sixth, fifth and fourth ribs are divided about 1 inch (2.5 cm.) lateral to the transverse vertebral processes.

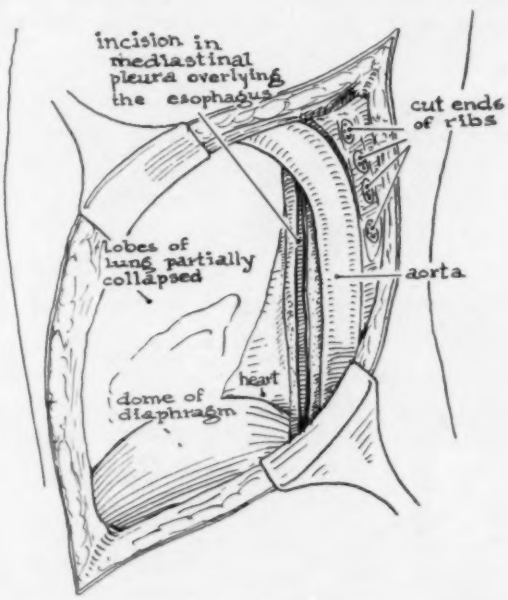


Fig. 2.—An incision is made in the infra-aortic mediastinal pleura, mesial to the aorta, in order to expose the esophagus.

care is exercised in ligating all small vessels, there will be surprisingly little loss of blood. The phrenic nerve is pushed to one side and usually remains out of the field of operation.

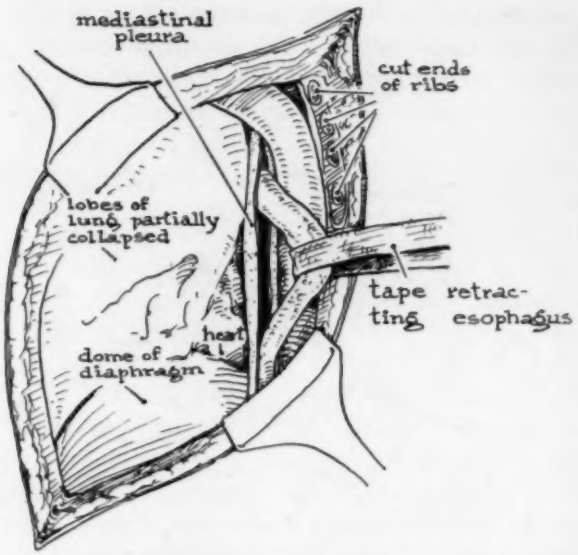


Fig. 3.—The esophagus is dissected from its attachment, and a tape is placed about it. Traction on the tape aids considerably in freeing the organ.

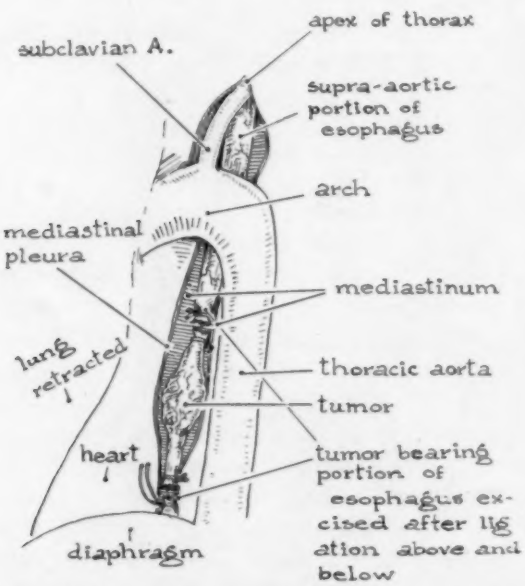


Fig. 4.—The mediastinal pleura above the arch is incised, and the esophagus is freed from the surrounding structures. The tumor-bearing portion of the organ, with a wide margin of normal tissue, is removed after double ligation and division.

Figures 1 to 4 have previously appeared (Surg., Gynec. & Obst. **70**:556-557 [Feb. 15] 1940) and are reproduced by permission of the editors.

Unless the neoplasm is hopelessly attached to the aorta, the operation should be continued. Not infrequently the tumor may be fixed to the mediastinal pleura on the right side. In my first case this complication was present. It was found necessary to excise a piece of the right pleura together with the tumor in order to effect a radical removal. This resulted in a bilateral pneumothorax, but the effect on the patient was barely discernible. The opening in the pleura was packed and later closed with a running stitch of catgut.

An incision is made in the mediastinal pleura above the arch of the aorta and is extended as far as the apex of the thorax (fig. 4). Pleural flaps are fashioned, and the underlying esophagus is freed from its surrounding attachments by careful finger dissection. The most shocking part of the operation occurs when the esophagus is separated from the posterior surface of the aortic arch. This maneuver must be executed with great care and with as little trauma as possible. The next step consists of division of the esophagus with the phenol cautery between heavy silk ligatures about 1 inch (2.5 cm.) above the diaphragm. The distal end is inverted into the stomach with two purse string sutures of silk. This may be reenforced by a layer of mattress sutures of chromic catgut. The esophagus is again divided between heavy silk ligatures above the neoplasm, and the tumor-bearing portion is removed (fig. 4). This is an important modification of the original Torek technic. Removal of the tumor immediately eliminates the infected portion of the esophagus and decreases greatly the possibility of contamination of the pleural cavity. It also obviates dragging the tumor-bearing area through the thoracic aperture into the cervical wound.

In order to avoid further the possibility of pleural contamination, which would occur if the silk ligature accidentally slipped off the esophageal stump, a rubber condom is placed over the end of the organ and sewn into place with a few fine silk sutures. Another heavy silk ligature tied securely about the condom gives added security.

The remaining segment of esophagus is brought to a position above the aortic arch by means of a curved clamp placed from above downward behind the arch and grasping the ligature surrounding the cut end of the organ (fig. 5).

After the esophageal stump has been brought to lie above the aortic arch, blunt dissection is continued into the root of the neck. The rubber-covered remains of the esophagus are then pushed upward into the neck, and the mediastinal pleura above the arch of the aorta is sutured to hold the organ in place (fig. 6). The mediastinal pleura below the

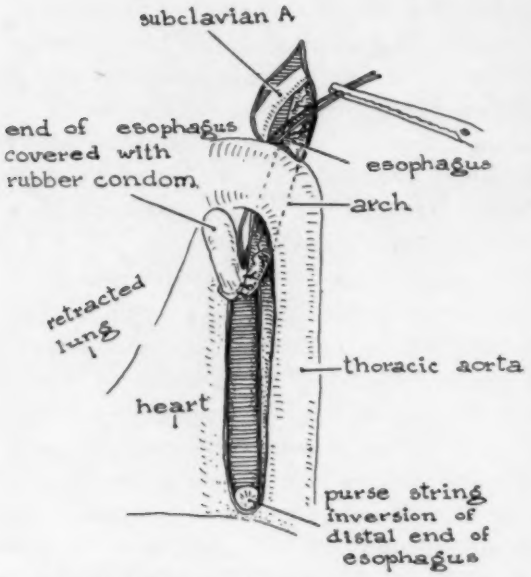


Fig. 5.—The divided end of the esophagus has been covered with a rubber condom, which is sutured to the esophageal wall. The distal end of the esophagus is inverted into the stomach. The mediastinal pleura is not sutured. The infra-aortic portion of the esophagus is brought to lie above the arch.

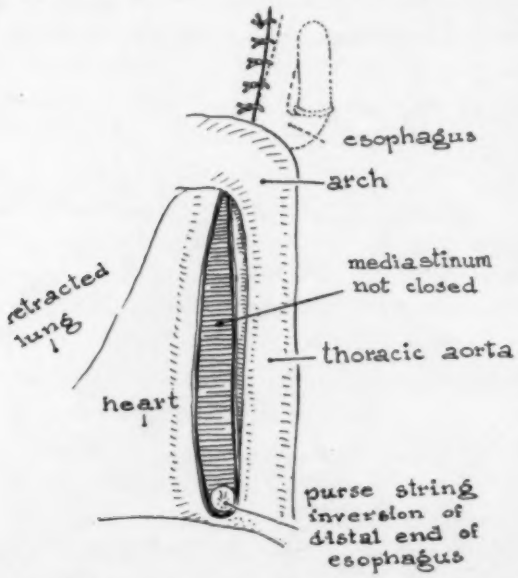


Fig. 6.—The mediastinum below the arch is left open to permit of drainage of the cellular tissues. The pleura above the arch is sutured in order to close off the neck from the pleural cavity. The rubber-covered end of the esophagus has been pushed into the neck.

arch should be left open. Free drainage of the cellular tissues in the mediastinum is important, and this can be obtained by not suturing the pleural flaps.

It is important, especially when the mediastinum is left open, to drain the thoracic cavity. Many intrathoracic complications may develop, and it is a comfort to have a vent for the first five or six days. For this purpose a stab wound is made posteriorly in one of the lower intercostal spaces, and a medium-sized soft rubber tube is inserted. This should be fastened to the chest wall by means of a suture passed through a cuff. When the patient is returned to bed, the outer end of the tube should be placed under water in a bottle beside the bed, insuring a closed system.

The thoracic wound is now ready for closure. The ribs are approximated by passing heavy chromic gut sutures around the seventh and eighth ribs. The divided thoracic musculature is next repaired with interrupted sutures of plain catgut, and the skin is closed in the usual way. After the dressing is applied and the chest securely strapped, the anesthetist should inflate the lungs until air bubbles no longer appear at the outer end of the intercostal tube, which has been placed in a basin of water.

The patient is then turned on his back. The neck and the anterior region of the chest are exposed and prepared. A complete clean instrument tray should be at hand for this stage. Gowns and gloves should be changed. An incision is made along the anterior edge of the left sternocleidomastoid muscle. This structure and the great vessels are retracted externally. The lateral thyroid vein is ligated and divided, and the lobe of the thyroid is retracted mesially (fig. 7) The cervical portion of the esophagus is easily identified and dissected free. A blunt hook is placed beneath it, and traction draws the remaining portion out of the root of the neck (fig. 7). A subcutaneous channel 1½ inches (3.7 cm.) in width is made, extending from the lower end of the cervical incision down to a point on the left side opposite the second rib (fig. 8). A transverse incision 1½ inches in length is made at this site and the esophagus is drawn through this tunnel. It is wise to have the esophagus project beyond this incision for an inch or two, because some necrosis of the end of the organ may take place. The esophageal wall is sutured to the cutaneous incision with interrupted silk stitches. The cervical incision is closed air tight, and a dressing is applied (fig. 9).

A word of warning about the blood supply of the esophageal stump is in order. This portion of the organ receives its nutrition from branches of the inferior thyroid artery. It is important, therefore, in conducting the cervical part of the operation, not to compromise the integrity of

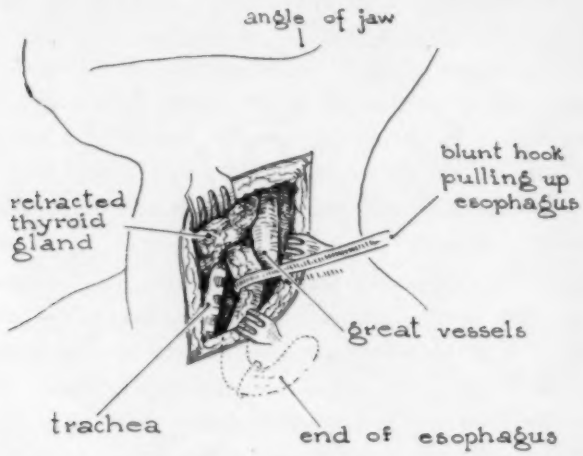


Fig. 7.—Second stage of the operation. The cervical portion of the esophagus is exposed, and the end of the organ, covered by the rubber envelope, as indicated in the dotted lines, is drawn out of the wound.

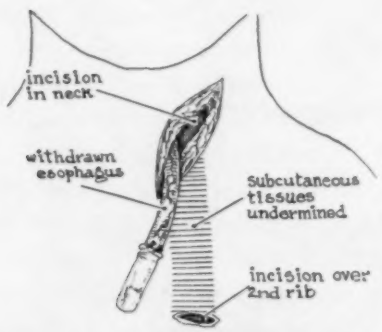


Fig. 8.—A subcutaneous channel is made, extending from the lower end of the cervical incision to about the region of the second rib.

this vessel. This requires careful retraction of the cervical incision, accurate hemostasis and the avoidance of rough blunt dissection.

The best insurance against shock on the operating table and also postoperatively, besides the already mentioned gentle operative technic, is a transfusion during the operation. To every patient operated on by me a transfusion of whole blood has been given on the operating table,

followed by a continuous intravenous drip of 5 per cent dextrose in saline solution. The latter should be continued postoperatively for two or three days. If the patient is elderly, edema of the lungs may develop if too much fluid is given by this method. The drip mechanism may be so regulated as to deliver to the patient between 2,000 and 3,000 cc. of fluid in twenty-four hours.

When the patient is returned to bed, he should be placed in a semi-recumbent position. The Gatch bed is ideal for this purpose. Measures should be instituted to combat shock. Although hypodermic medication, stimulating enemas and external heat have undoubted value, the most effective remedy is a transfusion of whole blood. Sufficient morphine should be given to relieve pain. In recent cases I have placed the patient in an oxygen tent immediately after operation, and the patients so treated apparently have had less difficulty with breathing

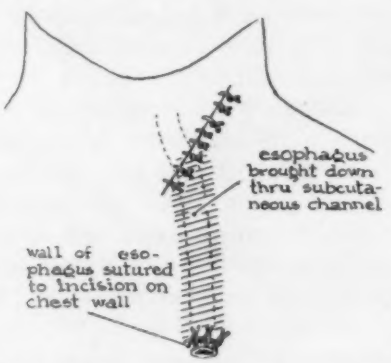


Fig. 9.—The esophagus is drawn through the channel so that its end projects beyond the cutaneous incision for a distance of 1 inch (2.5 cm.). The incision is sutured to the wall of the esophagus with interrupted stitches of fine silk.

Figures 5 to 9 have been published previously (Internat. Clin., vol. 1) and are reproduced by permission of the editors.

and fewer pulmonary complications. If inversion of the esophageal stump into the stomach has been satisfactory, fluid may be given by the gastrostomy tube. If inversion has not been satisfactory, it is wise to delay gastrostomy feeding until the sixth or seventh day. Regular feedings should be given as soon as the general condition of the patient permits.

The care of the esophageal stump is important. Beginning on the second day, the patient should be encouraged to swallow fluids, which must be caught in a basin. This cleanses the esophagus and helps to restore the act of deglutition. It is important to maintain good oral hygiene, and toward this end patients should be encouraged to chew gum.

The intrathoracic situation must be watched closely and may give the surgeon concern during the first week. Some indication of what is going on within the chest may be obtained from the temperature, the pulse rate, physical signs, roentgen examination of the chest and the amount and character of the material drained through the intercostal tube. If there is no dyspnea, if the temperature, pulse and physical signs are within normal limits and if the drainage diminishes each day, the tube may be removed permanently. If roentgen examination and the clinical course indicate the presence of empyema, surgical drainage will be indicated. It is important not to delay this step too long, because these patients, in view of their precarious condition, cannot stand infection for long.

The character of the material draining through the intercostal tube will indicate the progress of the intrathoracic situation. During the first two days the drainage is frankly sanguineous. After this period it gradually assumes a lighter color and decreases in amount until about the seventh or eighth day, when it usually stops completely. The tube may be removed soon afterward.

As soon as the patient's condition warrants it, usually about the end of the second week, the continuity of the esophagus should be established by inserting a rubber tube into the upper fistula and connecting it with the gastrostomy tube. The tube is held in place by carrying a tape around the patient's neck and attaching it to a safety pin which is inserted transversely into a rubber cuff placed about the tube about 2 inches (5 cm.) below its upper end. The patient is then encouraged to take all nourishment by mouth. In order to swallow solid food, he must thoroughly masticate it and mix it with more fluid than does the normal person, so that it will descend more easily through the tube into the stomach. It is hardly necessary to add that the rubber esophagus should be removed frequently and cleansed.

The care of the patient subsequent to his discharge from the hospital is chiefly that of experimenting with various sizes of rubber tubing until the rubber esophagus fits snugly into the fistula above. Leakage of ingested fluid from the upper end may be obviated by having the patient compress the orifice against the rubber tubing.

If infection has not been a major issue in the patient's convalescence, rapid improvement will be noted. There will usually be a rapid gain in weight and strength. The question of connecting the esophageal fistula and the gastrostomy opening by an antethoracic esophagoplasty should not be considered until there is reasonable assurance that the cancer has been eradicated. In most instances at least two years must elapse before such reconstructive work can be undertaken.

It has been my experience that tumors located between the arch of the aorta and the left main bronchus offer the greatest technical dif-

ficulties. Fixation of the esophageal wall to either structure takes place early. Even though this fixation may be inflammatory in origin and not due to direct neoplastic extension, actual separation of the structures from each other constitutes a formidable and difficult procedure. Because it is impossible accurately to differentiate inflammatory from neoplastic fixation, the surgeon should not abandon the lesion as inoperable and should make every effort to separate the structures. At the same time, I must admit that the highest recurrence rate will be found in this particular group of cases. In the next case of this type that presents itself, I plan to insert radium at the time of operation in the hope of eradicating any remaining cancer cells. I have operated on 3 patients with carcinoma in this unfavorable location. One died of a recurrence in the superior mediastinum a little over two years later; the second succumbed to local recurrence and generalized metastases one year later, and the third is alive six months after operation.

Discussion of this subject is not complete unless mention is made of the so-called "pull-through" operation described by Denk and popularized by Turner. This consists of a blind tunneling out of the thoracic portion of the esophagus by the combined abdominocervical route. My objection to this operation is that it is a blind procedure and that the surgeon has no way of telling whether the lesion is operable except by the sense of touch. The chance of injury of adjacent structures is obviously very real. In fact, Turner himself, the operation's greatest proponent, discussed with some misgivings his difficulties, such as tearing of the growth, injury to the pleura and postoperative hemorrhage. It seems to me that adequate exposure with direct vision of the field of operation is a sine qua non for radical surgical treatment of cancer.

A number of surgeons, notably Abel, Lotheissen, O'Shaughnessy and Ricard and Ballivet, have recommended a transpleural approach on the right side, claiming that the operation is technically easier. In order to expose the esophagus completely by this route it is necessary to ligate and divide the azygos vein. I have practiced this operation on the cadaver and am not convinced that it is easier than the transpleural operation on the left already described. I agree with Torek that the liver, diaphragm and pericardium interfere with adequate removal of the esophagus below the growth and render satisfactory inversion of the lower end into the stomach very difficult.

Carcinoma of the Lower Third of the Esophagus.—From the pathologic standpoint, cancers originating in the cardiac end of the stomach and secondarily involving the lower portion of the esophagus, that is, adenocarcinomas, should not be classed with the neoplasms originating in the esophagus itself, namely, the squamous cell tumors. The direction of lymphatic spread is quite different in each instance. Cancers

originating at the cardia spread to the gastrohepatic omentum and to the liver as well as to the mediastinum, often in the early stages of the disease, while esophageal tumors rarely extend to the lymph nodes below the diaphragm and remain purely local for some time. It will be seen, therefore, that carcinomas which arise from the esophageal mucosa will be more often amenable to resection. However, the same operation is applicable to cardial carcinomas, if preliminary abdominal exploration indicates operability.

When the growth is located in the distal third of the organ, every effort should be made to reestablish continuity of the esophagus and the stomach by some form of anastomosis. The decision to elect this operative plan will rest on three factors: (1) an accurate interpretation of the extent and location of the growth in the roentgenogram; (2) histologic examination of the biopsy specimen (squamous cell carcinoma), and (3) the distance between the upper incisor teeth and the upper level of the tumor as determined by the esophagoscopist. Any tumor located 38 cm. or more from the upper incisor teeth may be considered suitable for this operation, all other factors being equal.

For this type of lesion I do not recommend preliminary gastrostomy, because the adhesions in the upper part of the abdomen produced thereby will seriously hamper the surgeon in mobilizing the stomach. If a preliminary jejunostomy for feeding purposes is necessary, it is suggested that it be placed below the umbilicus.

All the evidence to date indicates that transthoracic resection with esophagogastrostomy in one stage is the procedure of choice for carcinoma of the lower part of the esophagus. It is based on the sound surgical principles of free exposure, mobilization of the involved viscera, preservation of blood supply, radical excision of the cancer-bearing focus with its associated lymph nodes and physiologic restoration of esophagogastric continuity by a layer suture anastomosis.

The operation, a modification of the original Sauerbruch and Fischer procedure, consists of a transthoracic approach on the left side, incision of the diaphragm, mobilization of the upper two thirds of the stomach, resection of the tumor-bearing area, performance of a careful suture anastomosis between the end of the esophagus and the anterior wall of the stomach in two layers and telescoping of the esophagus into the stomach by drawing the latter organ upward in a sleevelike manner around the esophagus in order to minimize any possible drag on the suture line.

Operative Technic.—The details of the operation are as follows: The patient is placed on his right side with the head resting on the abducted right arm and his back close to the edge of the operating table. The incision begins in the eighth interspace in the anterior axillary line

and is curved posteriorly, extending upward between the spine and the vertebral border of the scapula as far as the fourth rib (fig. 10). The thoracic musculature is divided in the line of the cutaneous incision. All bleeding points are carefully ligated before the thoracic cavity is entered. An incision is made in the eighth interspace, dividing the parietal pleura with the intercostal muscles. The eighth, seventh, sixth and fifth ribs are quickly divided near the spine, and the intercostal vessels are ligated with hemostatic sutures. Use of a rib spreader gives excellent exposure of the left side of the thoracic cavity. The inferior pulmonary ligament is divided, permitting collapse of the lower lobe. Complete pulmonary collapse should not be permitted to take place. This can be controlled by the anesthetist.

The growth is then palpated. Intimate fixation to the aorta or to the vertebral column renders the lesion inoperable. A vertical incision is made in the mediastinal pleura, mesial to the aorta, from the arch down to the diaphragm (fig. 11). The esophagus is identified and freed bluntly from the mediastinal tissues. It is important to ligate the small esophageal vessels, but care should be taken not to jeopardize the blood supply of the esophageal stump to be utilized for the anastomosis. Fixation to the right mediastinal pleura should not deter the surgeon. I have encountered this complication on three occasions and have in each instance excised a portion of the right pleura in order to effect a radical removal. The defect may be closed easily by utilizing the wall of the stomach after it has been mobilized.

An incision is then made in the left leaf of the diaphragm, extending from the esophageal hiatus radially outward toward the ribs (fig. 11) for a distance of 6 or 7 inches (15.2 to 17.7 cm.). The esophagocardiac junction is freed bluntly from its loose attachment to the diaphragm. The spleen and the fundus of the stomach quickly appear in the wound. Judicious retraction of the edges of the diaphragmatic opening and insertion of gauze packs will expose adequately the proximal half of the stomach. The left gastroepiploic vessels are ligated and divided, which permits the surgeon to enter the lesser sac. By inserting the left hand in the lesser sac and drawing the stomach downward, it is not difficult to isolate the gastric artery near its origin and to divide it after careful double ligation. Complete mobilization of the proximal half of the stomach is obtained after division of its ligamentous attachments at the upper part of the greater curvature. The stomach may then be drawn up into the thoracic cavity with ease (fig. 12).

During resection of the growth and the subsequent anastomosis, the entire thoracic cavity and wound must be protected from contamination. Long rubber-covered clamps are placed on the stomach side beyond the growth in an oblique direction from the upper portion of the greater

curvature toward the lesser curvature, and the stomach is divided with the phenol cautery. The opening in the stomach is closed in two layers, silk being used and care taken to ligate separately all bleeding points (fig. 13). A small rubber-covered noncrushing clamp is now placed

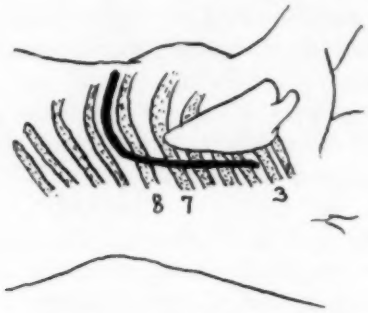


Fig. 10.—Resection and esophagogastrostomy. Incision is made in the eighth interspace. The eighth, seventh, sixth and fifth ribs are divided near the transverse vertebral processes.

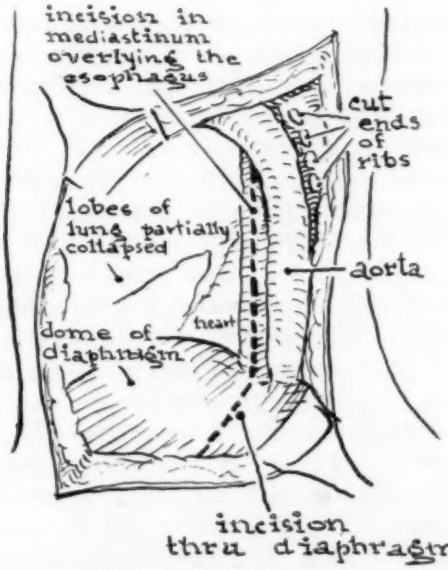


Fig. 11.—Incision is made in the mediastinal pleura. The diaphragm is split radially outward from the esophageal hiatus.

over the esophagus well above the tumor. A second crushing clamp is placed about ¾ inch (1.9 cm.) beyond this, and the esophagus is divided so as to leave at least ½ inch (1.2 cm.) of the organ projecting beyond the rubber-covered clamp. The specimen is removed.

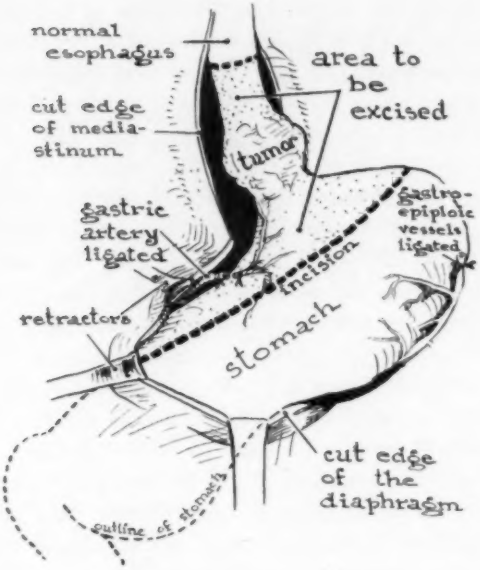


Fig. 12.—The tumor-bearing portion of the esophagus and the cardia have been separated from the mediastinum and the diaphragm. Mobilization of the stomach is accomplished by ligation and division of the gastric and left gastro-epiploic vessels.

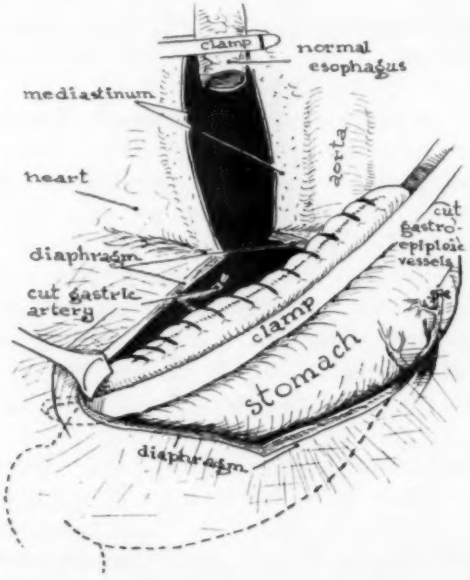


Fig. 13.—The stomach is resected in an oblique direction well beyond the growth and is closed with two layers of silk sutures. The esophagus is transected well above the cancer. A small rubber-covered clamp prevents soiling.

The upper end of the stomach in the region of the greater curvature is brought in approximation to the esophageal stump (fig. 14). A two layer suture anastomosis, end to side, is next performed between the end of the esophagus and the anterior wall of the stomach at a point about 2 inches (5 cm.) beyond its upper end (fig. 15). The inner layer is a continuous Connell suture of fine silk uniting the mucosa and the submucosa. Every stitch must be correctly placed. It is important to avoid strangulation of the tissues. The second layer is a continuous or interrupted Lembert suture of fine silk, joining the muscularis of the esophagus to the muscle and peritoneal layers of the stomach. In order to protect the suture line from longitudinal tension, the stomach is drawn upward like a sleeve over the suture line and is anchored in this position by interrupted sutures of fine silk which include the muscularis of the esophagus and the cut edges of the mediastinal pleura (fig. 16). The new anastomosis will usually be situated 1 or 2 inches (2.5 to 5 cm.) below the arch of the aorta, and it will be seen that approximately one half of the stomach now occupies an intrathoracic position.

The radial incision in the diaphragm is then closed with interrupted sutures of silk, care being taken not to make the opening through which the stomach emerges too small. To prevent herniation of more stomach into the chest, the edges of the diaphragmatic opening are sutured to the wall of the stomach (fig. 16). I cannot agree with Cattell and Marshall that it is imperative to crush the left phrenic nerve. In fact, I believe that there is real danger in this procedure. If the anastomosis has been made without tension, there need be no worry that it will be torn apart by movement of the diaphragm.

Through a small stab wound in the subjacent intercostal space a soft rubber tube is inserted for underwater drainage. The thoracic wound is then repaired in the usual manner by encircling the contiguous eighth and seventh ribs by heavy chromic sutures (fig. 17). The thoracic musculature is carefully repaired by layer, and the skin is closed with silk. The anesthetist then inflates the lungs, thus helping the escape of air in the pleural cavity through the intercostal drainage tube, the end of which has been placed under water. A snug dressing is next applied to support the severed ribs. If the right pleural cavity has been opened, the right side of the chest must be aspirated at the completion of the operation.

It has been both surprising and gratifying to see patients go through this extensive procedure with little evidence of shock. The pulse rate has varied between 80 and 126. The blood pressure readings have shown very little variation, a slight drop occurring during ligation of the gastric artery and mobilization of the stomach. In the last 2 cases I have been able to complete the operation in two hours and five minutes.

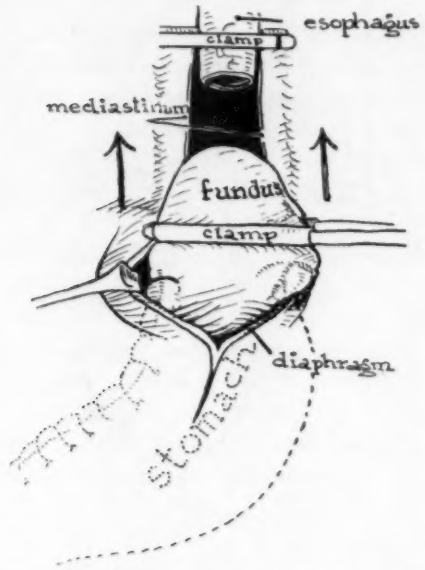


Fig. 14.—The stomach is drawn upward into the thoracic cavity and its upper end brought into position for anastomosis with the esophagus.

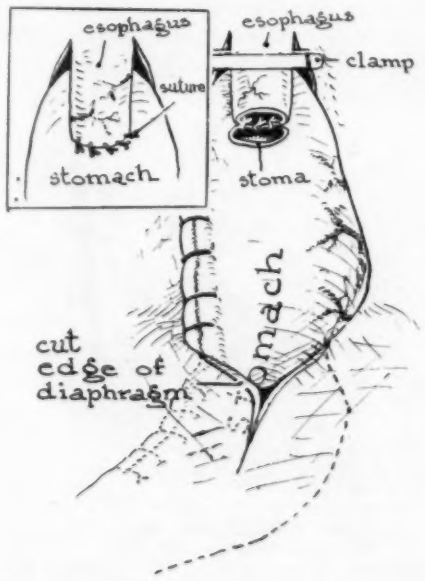


Fig. 15.—An end to side anastomosis in two layers is effected in the same manner as one performs gastroenterostomy. Silk sutures are used. Care should be taken to avoid strangulation of tissues. The insert shows the completed anastomosis.

Fig. 16.—The esophagus is telescoped into the stomach by drawing the stomach upward in a sleevelike manner. To maintain this position, the stomach is tacked down to the esophagus and also to the mediastinal pleura with interrupted stitches of silk. The remainder of the mediastinum is left open. The opening in the diaphragm is partly closed, the remaining edges being sutured to the wall of the stomach in order to prevent herniation.

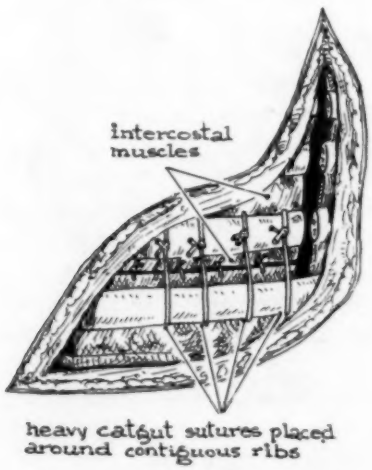


Fig. 17.—The thoracic wound is closed by encircling the contiguous eighth and ninth ribs with heavy catgut sutures.

Figures 10 to 17 have appeared previously (Surg., Gynec. & Obst. **70**:558-559 [Feb. 15] 1940) and are reproduced by permission of the editors.

POSTOPERATIVE CARE

Before undertaking this operation, it is most important to explain to the patient that he must not swallow for five or six days. The processes of repair at the suture line will be hastened by full cooperation of the patient in this respect. To supply fluids, use is made of the continuous intravenous drip of 5 per cent dextrose in physiologic solution of sodium chloride. It may be necessary to give another transfusion during the first day or two.

Small sips of water may be given on the fifth or sixth day. If nothing appears through the drainage tube, it is assumed that the suture line has healed and is intact. Increasing amounts of liquid are not given until about the sixteenth day, when custards, jellies, cereals, etc., are permitted. The diet is rapidly increased thereafter. Solid food should not be given until the third or fourth week.

The lumen at the site of anastomosis may diminish in caliber during the succeeding two or three months. If this happens, bougienage through an esophagoscope will become necessary. Such treatment should not be undertaken until there is reasonable assurance that the repair at the site of anastomosis is solid.

RESULTS

Up to the time of writing I have operated on 17 patients with carcinoma of the esophagus. Of this group, 6 were found to be inoperable, and 11 were treated by radical resection. The operability percentage was 64.7. In the group of 11 patients subjected to resection, 3 died postoperatively, a mortality of 27.2 per cent. One patient died of a tension pneumothorax on the right side. I believe that this death was due to an error in judgment and could have been prevented. The second patient died of a cerebral hemorrhage resulting in hemiplegia seventy-two hours after operation. At autopsy the intrathoracic situation was found to be satisfactory, and the anastomosis between the stomach and the esophagus was intact. There was no evidence of infection. The third patient, a physician of 54, died twelve hours after operation, of shock. The tumor in this instance was firmly attached to the diaphragm and the right pleura. The operation was unusually difficult and consumed almost four hours. Autopsy was not permitted.

In 7 of these cases the modified Torek operation was performed, with 1 death. In the remaining 4, resection with intrathoracic esophagogastrostomy was carried out. There were 2 deaths. The late results in the patients who survived operation are of considerable importance. Of the 8 survivors, 1 died of a recurrence in the superior mediastinum twenty-three months later; 1 died of coronary disease after three months, and 1 died of generalized metastases one year after operation. The 5

survivors are alive and well three and a half years, eleven months, seven months, six months and 1 month respectively after operation. In the second and third cases resection with esophagogastrostomy was performed.

In addition to the aforementioned group, I have operated on 10 additional patients with carcinoma of the cardia secondarily involving the lower part of the esophagus. Three of these were found to be operable, an operability rate of 30 per cent. In these 3 patients transthoracic resection with esophagogastrostomy was performed. There were 2 postoperative deaths. One patient, a woman of 72, in only fair condition, seemed slowly to disintegrate during the following three days. At postmortem examination the cause of death was not demonstrable. The suture line was intact, and there was no evidence of infection. The second patient died suddenly of a cerebral embolus on the third day. At autopsy the anastomosis was found to be intact, and there was no infection. The third patient is alive and well eight months after the operation. The cases comprising this group will form the basis of a subsequent paper.

In the group with cardial carcinoma it is important to find out whether the growth is resectable before opening the chest. The rapidity of growth of these tumors and the high incidence of inoperable lesions when the patients first consult the surgeon make this precaution advisable. Preliminary exploration of the upper part of the abdomen through a small left rectus incision will demonstrate the presence or absence of metastases to the liver, involvement of the lymph nodes and fixation of the growth to the surrounding structures. If the tumor seems operable, the chest may be opened and resection carried out. If inoperability is demonstrated, the incision is closed, or, if obstruction is a prominent factor, gastrostomy may be performed. By this plan an unnecessarily high mortality in the inoperable group will be avoided.

Rapid progress is being made in the surgical treatment of cancer of the esophagus, a disease considered until recently as hopeless. It is my firm conviction that in the near future surgical treatment will be the generally accepted method of handling this disease.

BIBLIOGRAPHY

Abel, A. L.: The Treatment of Cancer of the Esophagus (Hunterian Lecture), Brit. J. Surg. **14**:131-159, 1926.

Adams, W. E.; Escudero, L.; Aronsohn, H. G., and Shaw, M. M.: Resection of the Thoracic Esophagus: A Clinical and Experimental Study, J. Thoracic Surg. **7**:605-620, 1938.

——and Phemister, D. B.: Carcinoma of the Lower Thoracic Esophagus: Report of a Successful Resection and Esophagogastrostomy, ibid. **7**:621-632, 1938.

Beck, J. C., and Guttman, M. R.: Carcinoma of the Esophagus, Ann. Otol., Rhin. & Laryng. **41**:1133-1190, 1932.

Bengolea, A. J.: Radical Resection of the Esophagus for Carcinoma, Surg., Gynec. & Obst. **29**:413-415, 1919.

Bessesen, D. H., and Bessesen, A. N., Jr.: Surgical Treatment of Carcinoma of the Thoracic Esophagus, with Report of a Case, Am. J. Surg. **12**:437-442, 1931.

Billroth, T.: Ueber die Resection des Esophagus, Arch. f. klin. Chir. **13**:65-69, 1871.

Bircher, E.: Operative Heilung eines Karzinomes am Übergang des Oesophagus in die Kardia, Cor.-Bl. f. schweiz. Aerzte **48**:467-478, 1918.

Bricker, E. M., and Graham, E. A.: The Inhibitory Effect of Sulfanilamide on Wound Healing, J. A. M. A. **112**:2593-2594 (June 24) 1939.

Brunn, H., and Stephens, H. B.: Carcinoma of the Thoracic Esophagus: A Report of the Successful Removal in One Case, J. Thoracic Surg. **7**:38-42, 1937.

Buchbinder, W. C.: Carcinoma of the Esophagus with Especial Reference to Site, Am. J. M. Sc. **170**:496-499, 1925.

Bunnell, S.: Esophagectomy, Surg., Gynec. & Obst. **34**:408-410, 1922.

Cain, A., and Solomon, I.: Contribution à l'étude de la radiosensibilité du cancer de l'œsophage, Presse méd. **45**:334-336, 1937.

Churchill, E. D.: Esophageal Surgery, Surg., Gynec. & Obst. **60**:417-423, 1935.

Clairmont, P.: Zur radikal Operation des Oesophaguskarzinoms, Zentralbl. f. Chir. **51**:42-46, 1924.

Cleminson, F. J.: Thoracotomy in the Treatment of Malignant Disease of the Esophagus by Radon, J. Laryng. & Otol. **44**:577-583, 1929.

Crump, A., and Kasabach, H.: A Report of a "Proved Cured Case" of Squamous Cell Epithelioma of the Esophagus Treated by Intraesophageal and External Irradiation, J. Thoracic Surg. **5**:157-168, 1935.

Decker, H. R.: Report of a Case of Esophagogastrostomy for Carcinoma of the Esophagus, J. Thoracic Surg. **5**:143-156, 1935.

Demel, R.: Das Karzinom der Speiseröhre und des Magens, Wien. med. Wchnschr. **82**:178-182, 1932.

Denk, W.: Zur Radikaloperation des Oesophaguskarzinoms, Zentralbl. f. Chir. **40** (pt. 2):1065-1068, 1913.

Dufourmentel, L.: Le traitement curatif du cancer de l'œsophage, Paris méd. **2**:87-89, 1930.

Edwards, A. T.: Extirpation of the Esophagus for Carcinoma, J. Laryng. & Otol. **51**:281-292, 1936.

Eggers, C.: Resection of the Thoracic Portion of the Esophagus for Carcinoma: Report of a Successful Case, Arch. Surg. **10**:361-373 (Jan., pt. 2) 1925.

Carcinoma of the Thoracic Portion of the Esophagus, Surg., Gynec. & Obst. **50**:630-634, 1930.

Resection of the Thoracic Portion of the Esophagus for Carcinoma, ibid. **52**: 739-746, 1931.

Experiences with Carcinoma of the Esophagus, J. Thoracic Surg. **2**:229-246, 1933.

Treatment of Carcinoma of the Esophagus, Surg., Gynec. & Obst. **63**:54-65, 1936.

Upper Esophagostomy: Its Indications and Uses, J. Thoracic Surg. **7**:633-648, 1938.

Emery, E. S., Jr.: The Relative Value of Symptoms Versus the X-Ray and Esophagoscope in the Early Diagnosis of Carcinoma of the Esophagus, New England J. Med. **210**:420-421, 1934.

Farrell, J. T., Jr.: Roentgen Diagnosis of Carcinoma of the Esophagus, Radiology **14**:282-295, 1930.

Fischer, A. W.: Erfolgreiche Entfernung eines Krebses der Speiseröhre im mittleren Brustteil nach dem abdominocollaren Durchzugsverfahren, Arch. f. klin. Chir. **189**:498-512, 1937.

Fischer, H.: Surgical Treatment of the Esophagus, Arch. Surg. **6**:256-273 (Jan., pt. 2) 1923.

Esophageal Implantation into Stomach After Intrathoracic Resection of the Esophagus for Carcinoma: A New Method, ibid. **12**:241-246 (Jan., pt. 2) 1926.

Friedenwald, J.; Zinn, W. F., and Feldman, M.: Cancer of the Esophagus, Am. J. M. Sc. **174**:609-623, 1927.

Garlock, J. H.: The Surgical Treatment of Carcinoma of the Thoracic Esophagus, with a Report of Three Successful Cases, Surg., Gynec. & Obst. **66**:534-548, 1938.

The Surgical Treatment of Carcinoma of the Thoracic Esophagus, Internat. Clin. **1**:28-45, 1939.

The Surgical Treatment of Carcinoma of the Esophagus, Surg., Gynec. & Obst. **70**:556-569, 1940.

Gohrbandt, E.: Zur Operation des intrathorakalen Oesophaguskarzinoms, Zentralbl. f. Chir. **54**:26-28, 1927.

Die abdominale und kolloabdominale Resektion des intrathorakal gelegenen Oesophaguscarcinoms, Arch. f. klin. Chir. **148**:528-535, 1927.

Greene, D. C.: Treatment of Carcinoma of the Esophagus by Radiation, Am. J. Roentgenol. **12**:471-474, 1924.

Guisez, J.: Plusieurs cas de cancers de l'œsophage traités avec succès par la radiumthérapie, Bull. et mém. Soc. de chir. de Paris **22**:751, 1930.

Cancer de l'œsophage traité par la radiumthérapie, récidive au bout de vingt-six ans, ibid. **28**:564, 1936.

von Hacker, V., and Lotheissen, G.: Die Chirurgie der Speiseröhre, in Garré, C.; Küttner, H., and Lexer, E.: Handbuch der praktischen Chirurgie, ed. 5, Stuttgart, Ferdinand Enke, 1920, vol. 2.

Chirurgie der Speiseröhre, in von Bruns, P.: Neue deutsche Chirurgie, Stuttgart, Ferdinand Enke, 1926, vol. 34.

Hedblom, C. A.: Combined Transpleural and Transperitoneal Resection of the Thoracic Esophagus and the Cardia for Carcinoma, Surg., Gynec. & Obst. **35**:284-287, 1922.

Helsley, G. F.: The Metastasizing Tendency of Esophagus Carcinoma, Ann. Surg. **77**:272-275, 1923.

Heuer, G. J.; Andrus, W. de W., and Bell, H. G.: The Experimental Transplantation of the Diaphragm as an Adjunct in the Treatment of Lesions at the Lower End of the Esophagus: Application of the Method in a Case of Carcinoma of the Cardiac End of the Esophagus in Man, Ann. Surg. **81**: 273-286, 1925.

Hickey, P. M.: Cancer of the Lower End of the Esophagus and Cardiac End of the Stomach, J. A. M. A. **93**:1356-1358 (Nov. 2) 1929.

Hill, W.: Radium Therapy in Cancer of the Esophagus: The Results of Fifteen Years' Experience, Brit. M. J. **2**:1196-1197, 1924.

Hoffman, F. L.: Carcinoma of the Esophagus, New England J. Med. **211**:769-772, 1934.

Holmes, G. W., and Schatzki, R.: Examination of the Mucosal Relief as a Diagnostic Aid in Diseases of the Gastrointestinal Tract, Am. J. Roentgenol. **34**:145-167, 1935.

Hoover, W. B.: Carcinoma of the Esophagus, S. Clin. North America **18**:633-641, 1938.

Ingebrigtsen, R.: Résection du cardia cancéreux, Acta chir. Scandinav. **73**: 43-45, 1933.

Pharyngo-Esophageal Cancer: Resection, Suture and Primary Healing, Norsk mag. f. lægevidensk. **94**:500-504, 1933.

Resection of Cardia and Lower Portion of Esophagus in Cases of Cancer, ibid. **9**:479-486, 1936.

Jackson, C.: Carcinoma and Sarcoma of the Esophagus: A Plea for Early Diagnosis, Am. J. M. Sc. **169**:625-648, 1925.

Carcinoma and Sarcoma of the Esophagus, South. Surgeon **4**:1-11, 1935.

Janeway, H. H., and Green, N. W.: Cancer of the Esophagus and Cardia, Ann. Surg. **52**:67-82, 1910.

Keefer, C. S.: The Pleural and Pulmonary Complications of Carcinoma of the Esophagus, Ann. Int. Med. **8**:72-81, 1934.

King, E. S. J.: Surgical Treatment of Carcinoma of the Thoracic Esophagus, M. J. Australia **1**:399-401, 1936.

Esophagectomy for Carcinoma of the Thoracic Esophagus, Brit. J. Surg. **23**:521-529, 1936.

A Case of Esophagectomy (Torek's Operation) for Carcinoma, and Esophagoplasty, Australian & New Zealand J. Surg. **6**:307-316, 1937.

Küttner, H.: Gelungene Ösophagusresektion wegen Karzinoms vom Halse aus mit breiter Eröffnung der Pleura und intrapleuraler Versorgung des unteren Ösophagusstumpfes, Zentralbl. f. Chir. **48** (pt. 1):846, 1923.

Leven, N. L., and Bowers, W. F.: Carcinoma of Esophagus, Staff Meet. Bull., Hosp. Univ. Minnesota **9**:126-132, 1938.

Lewis, F. O.: Resection of the Cervical and Upper Thoracic Esophagus for Carcinoma: Report of a Case, Ann. Otol., Rhin. & Laryng. **43**:1192-1197, 1934.

Lilienthal, H.: Carcinoma of the Thoracic Esophagus: Successful Resection, Ann. Surg. **74**:116-120, 1921.

Lotheissen, G.: Zur Radikaloperation des Speiseröhrenkrebses, Arch. f. klin. Chir. **131**:200-225, 1924.

Die Resektion der Speisröhre, Wien. med. Wchnschr. **79**:618-619, 1929.

Behandlung der organischen Stenosen des Oesophagus und des Kardiospasmus, München. med. Wchnschr. **81**:41-45, 1934.

McGibbon, J. E. G.: The Clinical Manifestations of the Spread of Carcinoma of the Esophagus Observed During Life, Brit. J. Surg. **24**:86-104, 1936.

Marshall, S. F.: Carcinoma of the Esophagus: Successful Resection of Lower End of Esophagus with Re-Establishment of Esophageal Gastric Continuity, S. Clin. North America **18**:643-648, 1938.

Marwedel, G.: Die Aufklappung des Rippenbogens zur Erleichterung operativer Eingriffe im Hypochondrium und im Zwerchfellkuppelraum, Zentralbl. f. Chir. **30**:938-941, 1903.

Mathews, R. W., and Schnadel, T. G.: Primary Esophageal Carcinoma, with Especial Reference to a Nonstenosing Variety: A Clinicopathologic Study Based on One Hundred and Eight Necropsies, J. A. M. A. **105**:1591-1595 (Nov. 16) 1935.

Meyer, W.: The Surgical Treatment of Cancer of the Esophagus, M. Rec. **83**: 888-889, 1913.

Further Experience with Resection of the Esophagus for Carcinoma, Surg., Gynec. & Obst. **20**:162-170, 1915.

The Early Diagnosis of Cancer of the Esophagus, Am. J. Surg. **29**:252-255, 1915.

von Mikulicz, J.: Ein Fall von Resection des carcinomatösen Oesophagus mit plastischem Ersatz des excidirten Stückes, Prag. med. Wchnschr. **11**:93-94, 1886.

Miller, R. T., Jr., and Andrus, W. de W.: Experimental Surgery of the Thoracic Esophagus, Bull. Johns Hopkins Hosp. **34**:109-114, 1923.

Moersch, H. J.: Diagnosis and Treatment of Carcinoma of the Esophagus, Minnesota Med. **12**:582-586, 1929.

Treatment of Carcinoma of the Esophagus by Means of Surgical Diathermy, J. Thoracic Surg. **7**:43-47, 1937.

Muir, E. G.: Resection of the Lower Esophagus and Cardia, Lancet **2**:75-77, 1936.

New, G. B., and Vinson, P. P.: Metastasis from Symptomless Cancer of Esophagus, M. J. & Rec. (supp.) **120**:xix-xx, 1924.

Nissen, R.: Die transpleurale Resektion der Kardia, Deutsche Ztschr. f. Chir. **249**:311-316, 1937.

Ohsawa, T.: The Surgery of the Esophagus, Arch. f. jap. Chir. **10**:605-700, 1933.

Orton, H. B.: Lateral Transthyroid Pharyngotomy: Trotter's Operation for Malignant Conditions of the Laryngopharynx, Arch. Otolaryng. **12**:320-338 (Sept.) 1930.

Resection of the Entire Thoracic Esophagus for Carcinoma, Ann. Otol., Rhin. & Laryng. **45**:28-32, 1936.

O'Shaughnessy, L., and Raven, R. W.: Surgical Exposure of the Esophagus, Brit. J. Surg. **22**:365-377, 1934.

Pancoast, H. K.: Roentgenology of the Pharynx and Upper Esophagus, Am. J. Cancer **17**:373-395, 1933.

Pilcher, R.: Carcinoma of the Cervical Esophagus, Lancet **1**:73-76, 1937.

Pirie, A. H.: Treatment of Carcinoma of Esophagus by Deep X-Ray Therapy, Am. J. Roentgenol. **10**:459-461, 1923.

Radford, A.: Excision of the Thoracic Esophagus for Carcinoma, South African M. J. **11**:498-499, 1937.

Raven, R. W.: Cancer of the Esophagus, St. Barth. Hosp. Rep. **64**:181-194, 1931.

Rehm: Operationen an dem Brustabschnitt der Speiseröhre, Verhandl. d. deutsch. Gesellsch. f. Chir. **27**:448-470, 1898.

Ricard, A., and Ballivet: Chirurgie radicale du cancer de l'œsophage thoracique. Possibilité et difficulté d'établir le diagnostic précoce indispensable, Lyon méd. **160**:569-577, 1937.

Œsophagectomie totale pour cancer de l'œsophage thoracique, Lyon chir. **35**: 94-99, 1938.

Rutherford, R.: Excision of the Esophagus for Carcinoma, Brit. J. Surg. **22**: 340-342, 1934.

Saint, J. H.: Surgery of the Esophagus, Arch. Surg. **19**:53-128 (July) 1929.

Sauerbruch, F.: Die Chirurgie des Brustteils der Speiseröhre, Beitr. z. klin. Chir. **46**:405-494, 1905.

Ueber die Indikationen zur Resektion des Brustabschnittes der Speiseröhre, Deutsche Ztschr. f. Chir. **98**:113-125, 1909.

Chirurgie der Brustorgane, Berlin, Julius Springer, 1925, vol. 2, pp. 607-608.

Seiffert, A.: Operation des Oesophaguscarcinoms auf endoskopischem Wege, Ztschr. f. Hals-, Nasen- u. Ohrenh. **24**:585-587, 1929.

Resultate der endoskopischen Operation des Oesophaguscarcinoms, Arch. f. klin. Chir. **181**:512-514, 1935.

Sherman, J.: Carcinoma of the Esophagus: A Report of Fifty Cases, Am. J. M. Sc. **175**:79-84, 1928.

Souttar, H. S.: Treatment of Carcinoma of the Esophagus, Based on One Hundred Personal Cases and Eighteen Post-Mortem Reports, Brit. J. Surg. **15**:76-94, 1927.

Cancer of the Esophagus, Brit. M. J. **2**:797-800, 1934.

Tasche, L. W.: Carcinoma of the Esophagus, Wisconsin M. J. **31**:523-527, 1932.

Teperson, H. I.: Treatment of Carcinoma of the Esophagus with Radon Implants: A Description of an Improved Implanter, Am. J. Roentgenol. **28**:229-235, 1932.

Torek, F.: The First Successful Case of Resection of the Thoracic Portion of the Esophagus for Carcinoma, Surg., Gynec. & Obst. **16**:614-617, 1913.

Carcinoma of the Thoracic Portion of the Esophagus: Report of a Case in Which Operation Was Done Eleven Years Ago, Arch. Surg. **10**:353-360 (Jan., pt. 2) 1925.

Carcinoma of Larynx, Pharynx, Trachea and Esophagus, Ann. Surg. **84**:889-890, 1926.

Surgery of the Esophagus, in Nelson Loose-Leaf Living Surgery, New York, Thomas Nelson & Sons, 1927, vol. 4, p. 212.

The Causes of Failure in the Operative Treatment of Carcinoma of the Esophagus, Ann. Surg. **90**:496-506, 1929.

Truesdale, P. E.: Esophagectomy for Carcinoma of the Upper Esophagus with Lessons Derived from an Operative Failure, J. Thoracic Surg. **3**:235-240, 1934.

Turner, A. L.: Carcinoma of the Post-Cricoid Region (Pars Laryngea Pharyngis) and Upper End of the Esophagus, Edinburgh M. J. **25**:345-362, 1920.

Turner, G. G.: Henry Jacob Bigelow Lecture: Some Experiences in the Surgery of the Esophagus, New England J. Med. **205**:657-674, 1931.

Personal Experiences in the Surgery of the Lower Esophagus, Verhandl. d. 9. Kong. d. internat. Gesellsch. f. Chir. **1**:725-740, 1932.

Excision of the Thoracic Esophagus, with Construction of an Extrathoracic Gullet, Lancet **2**:1315-1316, 1933; New England J. Med. **210**:947-948, 1934.

Recent Advances in the Treatment of Carcinoma of the Esophagus from the Surgical Aspect, J. Laryng. & Otol. **49**:297-312, 1934.

Excision of the Thoracic Esophagus for Carcinoma, Lancet **2**:1293-1294, 1934.

Carcinoma of the Esophagus: The Question of Its Treatment by Surgery (Bradshaw Lecture), ibid. **1**:67-72 and 130-134, 1936.

——and Knight, G. C.: Surgery of the Esophagus, Tr. M. Soc. London **59**:171-181, 1936.

——and others, in Discussion on Recent Advances in the Treatment of Carcinoma of the Esophagus, from the Surgical and Radiological Aspects, Proc. Roy. Soc. Med. **27**:355-380, 1934.

Unger, E.: Zur Chirurgie des intrathorakalen Oesophaguscarcinoms, Arch. f. klin. Chir. **106**:31-47, 1915.

Vinson, P. P.: Carcinoma of the Esophagus, Am. J. M. Sc. **166**:402-414, 1923.

The Treatment of Carcinoma of the Esophagus, Surg., Gynec. & Obst. **62**:840-842, 1936.

——and Moersch, H. J.: Dilation versus Gastrostomy as a Palliative Treatment of Carcinoma of the Esophagus, J. A. M. A. **84**:658-659 (Feb. 28) 1925.

Voelcker: Ueber Extirpation der Cardia wegen Carcinoms, Verhandl. d. deutsch. Gesellsch. f. Chir. **37**:126-129, 1908.

Watson, W. L.: Carcinoma of the Esophagus; Case Reports Illustrating two Methods of Treatment, S. Clin. North America **13**:444-448, 1933.

Carcinoma of the Esophagus, Surg., Gynec. & Obst. **56**:884-897, 1933.

Carcinoma of the Esophagus, Internat. J. Med. **47**:204-205, 1934.

Cancer of the Esophagus, New York State J. Med. **36**:1615-1619, 1936.

Wilensky, N.: Ueber Ösophagektomie und Ösophagoplastik bei hochsitzenden Ösophaguskarcinomen, Wien. klin. Rundschau **28**:55-59, 1914.

Woodman, E. M.: The Insertion and Use of Radon in Cancer of the Esophagus, J. Laryng. & Otol. **44**:584-592, 1929.

Treatment of Carcinoma of the Esophagus, Brit. M. J. **2**:290-292, 1931.

Wright, A. J.: The Technique of the Use of Radium in Malignant Disease of the Upper Air and Food Passages, J. Laryng. & Otol. **44**:365-371, 1929.

Zaaijer, J. H.: Erfolgreiche transpleurale Resektion eines Cardiacarcinoms, Beitr. z. klin. Chir. **83**:419-430, 1913.

Surgery of the Esophagus and Lungs, Lancet **1**:909-914, 1929.

Beitrag zur operativen Behandlung des intrathorakalen Oesophaguscarcinoms, Arch. f. klin. Chir. **162**:693-694, 1930.

On Surgery of the Esophagus, Verhandl. d. 9. Kong. d. internat. Gesellsch. f. Chir. **1**:485, 1932.

Zuppinger, A.: Die Behandlung der Oesophaguskarzinome, Ergebn. d. med. Strahlenforsch. **7**:389-456, 1936.

MALIGNANT TUMORS OF HERNIAL SACS

LEO M. ZIMMERMAN, M.D.
AND
HAROLD LAUFMAN, M.D.
CHICAGO

The sacs of inguinal hernias have been known to contain almost every type of normal and diseased abdominal viscus. Malignant tumors of organs contained in the sacs have been described, and, less frequently, secondary involvement of the tissues of the sac by extension or metastasis has been noted. Primary malignant tumors of the tissues of hernial sacs, however, are extremely infrequent. Baretz [1] reported an adenocarcinoma of a hernial sac, but it is obvious that a tumor of this type could not have arisen primarily from any but an epithelial structure and could not, therefore, have been primary in the sac. Unfortunately, postmortem examination was not done. Pagliani [2] reported a benign tumor, a fibromyxoma, which was primarily in the sac and which had been diagnosed preoperatively as a strangulated hernia. The only case encountered in the literature of the past two decades of primary malignant tumor of the sac of an inguinal hernia was cited by Richard [3] from a dissertation by Roessle, in which postmortem examination disclosed a fist-sized tumor of a hernial sac with metastases to the inguinal and para-aortic lymph nodes and in which a diagnosis of malignant mesothelioma was made.

Because of the rarity of malignant neoplasms involving hernial sacs, we are briefly reporting 3 cases, in 1 of which we have complete data, including postmortem observations. In the other 2 cases, while a diagnosis of malignant mesodermal tumor of the excised sac tissues was made, the data are somewhat equivocal. One of the patients is still alive and free from recurrence, and in the case of the other permission for autopsy could not be obtained. Each of these patients presented a different type of tumor, but all the growths were sarcomas, that is, were malignant neoplasms of mesodermal structure. All were rather bizarre forms, and positive histologic identification was difficult.

Read before the Chicago Surgical Society, March 1, 1940.

From the Departments of Surgery of the Michael Reese Hospital and the Chicago Memorial Hospital.

1. Baretz, L. H.: Carcinoma of Hernial Sac, Am. J. Surg. **7**:692-693, 1937.
2. Pagliani, F.: Tumore del sacca erniario, Bull. d. sc. med., Bologna **109**:108-119, 1937.
3. Richard, M.: Ueber Tumorbildung in Hernien, Deutsche Ztschr. f. Chir. **226**:146-152, 1930.

In each of the cases to be described the patient was operated on with a diagnosis of incarcerated inguinal hernia, and the presence of a malignant growth was not suspected until microscopic examination of the excised sac tissues was made. It must be understood that hernial sacs consist of peritoneum and subperitoneal areolar tissue. These layers are continuous with analogous layers of the abdominal wall. In 2 of these cases the malignant process was more or less diffuse; there is no way of knowing specifically in which portion of these layers the tumors arose, and we do not contend that they necessarily involved the hernial tissues first. The tumor in the third case was apparently limited to the sac of the hernia.

REPORT OF CASES

CASE 1.—R. P., a boy 11 weeks old, was admitted to the Michael Reese Hospital on Sept. 5, 1938, with a diagnosis of strangulated left inguinal hernia. The scrotal swelling had been present for eight days, and vomiting, restlessness and abdominal distention had been noted intermittently for four days. On the day of admission an attempt had been made to reduce the hernia, and the child had gone into shock.

Because of a tense, fluctuant swelling in the left side of the scrotum, which extended up into the inguinal region, and abdominal distention, operation was done. Inguinal incision disclosed a thickened and edematous hernial sac which was filled with fluid, but contained no viscus. As the ring was widened, there was a profuse flow of clear, straw-colored ascitic fluid from within the abdomen. Because the fluid was not bloody, laparotomy for possible reduced gangrenous bowel was not done. The sac was removed and the ring closed.

The immediate postoperative course was satisfactory. Subsequently a febrile course supervened, which subsided with the drainage of clear ascitic fluid between the sutures of the inguinal incision. The pathologist's diagnosis, returned at that time, was highly malignant tumor of mesenchymal origin. The fever recurred on the seventh postoperative day, and the course was then progressively downhill until death occurred, on the ninth day after operation.

Autopsy observations were relatively meager. There was fluid in the abdomen and in both pleural cavities, compressing the lungs. Scattered, flat elevations were seen distributed over the intestine and on the pleural surface of the diaphragm. There was, curiously, no other involvement of the parietal peritoneum than that of the hernial sac. When sectioned, the infiltrations were just beneath the serosal surfaces and did not involve the deeper layers of the intestinal wall.

Microscopic examination of the excised hernial sac, as well as of the serosal plaques and mesenteric lymph nodes, revealed large cells with distinct vesicular nuclei, each containing a clearcut nucleolus. Occasional giant cells were seen (fig. 1). The pathologist's diagnosis was primary peritoneal malignant tumor, a so-called mesothelioma of the peritoneum.

CASE 2.—M. M., a 64 year old white man, was operated on on Feb. 6, 1939, because of a right inguinal hernia which had been present for twenty years. For the past three months there had been symptoms of incomplete intestinal obstruction and considerable loss of weight. The patient had attempted to wear a truss, but as the hernia was irreducible the truss merely aggravated the pain.

At operation the sac was greatly thickened and infiltrated and was firmly adherent to the contained intestine. The bowel and its mesentery were greatly thickened, and the serosal surfaces were roughened. There were numerous transparent nodules on the surface which appeared to consist of inspissated fibrin. As the internal ring was dilated, a generous flow of ascitic fluid from the abdominal cavity appeared. A finger inserted into the peritoneal cavity encountered nodularity of the serosal surfaces comparable to that present in the sac.

The bowel was freed from the sac and reduced. At one point the adhesion was so dense that separation seemed perilous, and a button of thickened sac was left attached to the surface of the bowel. The remainder of the sac was removed and the canal repaired. The postoperative course was uneventful.

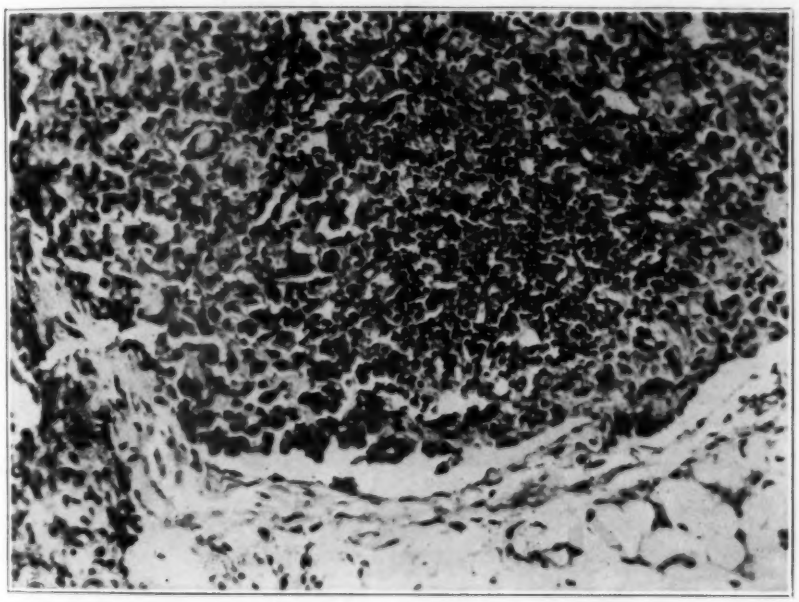


Fig. 1.—Metastasis to a lymph node. The section contains many lymphocytes which are part of the normal lymph node and an infiltration of large cells showing a distinct vesicular nucleus with a clearcut nucleolus. These cells are seen in the marginal sinus and invade the lymph node proper. Occasionally a multinucleated giant cell is seen. [Note: The primary tumor apparently arose in the peritoneum, a so-called mesothelioma of the peritoneum.]

Rather to our consternation, the pathologist again reported a malignant tumor. At first this was thought to be metastatic from the colon because of the presence of mucoid cells, but subsequent detailed study indicated that the growth was probably a malignant tumor of mesodermal origin, and a presumptive diagnosis of liposarcoma was made. The patient failed rapidly despite palliative irradiation and died six months after operation. Unfortunately, consent for autopsy could not be obtained.

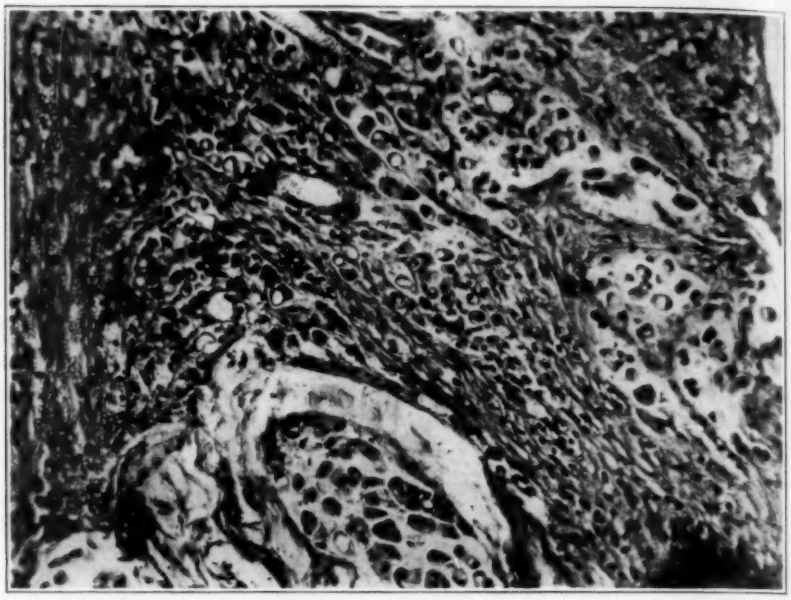


Fig. 2.—Accumulations of large cells arranged in groups. Many of the cells show a signet-shaped nucleus and a lightly staining (empty) cytoplasm. These cells invade the tissue. This was probably a very malignant liposarcoma. However, the possibility of its being part of metastatic mucinous adenocarcinoma of the large intestine could not be ruled out.

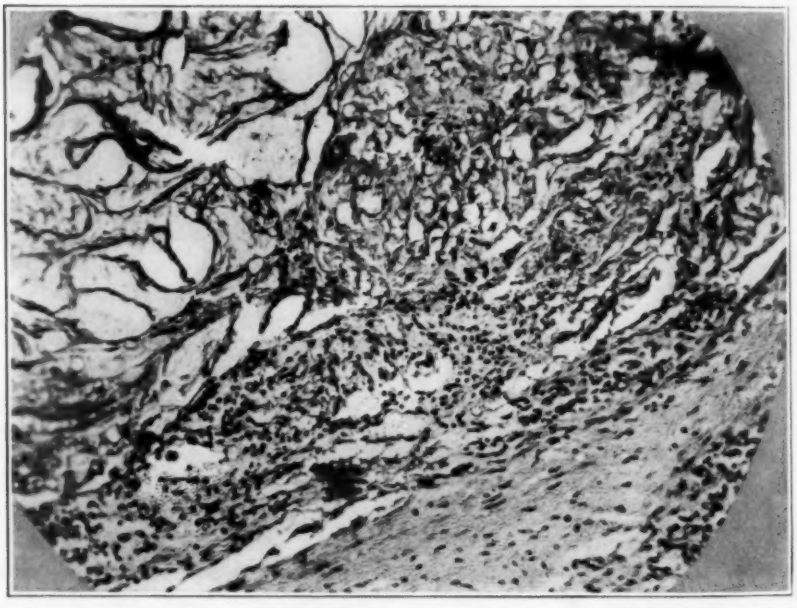


Fig. 3.—Degenerated connective tissue fibers separated by large empty spaces, with very few cellular elements in this region. The periphery shows a rather rich infiltration with lymphocytes. It is possible that this was a connective tissue tumor of the type of a fibromyxosarcoma. It is not likely that the empty spaces consisted of a foreign body of the type of paraffin, though that must be taken into consideration. There was no evidence of epithelial structures in this tumor.

The pathologist's report stated: "The tissue is invaded by accumulations of large cells which are arranged in groups. Many of the cells have signet-shaped nuclei and a lightly staining or empty cytoplasm. This is probably a very malignant liposarcoma. However, the possibility of its being part of a metastatic mucinous adenocarcinoma of the large intestine cannot be ruled out" (fig. 2).

CASE 3.—This case is included in this report through the courtesy of Dr. Harry Stimson, who operated on the patient, a 60 year old man, on Feb. 21, 1939, at the Chicago Memorial Hospital, because of an incarcerated left inguinal hernia of about two weeks' duration. The sac was found to be of the indirect type and was attached to the cord. Its walls were thick and edematous. The changes were limited to the tissues of the sac of the hernia and stopped abruptly at the internal ring. The sac was excised and the hernia repaired. The patient had an uneventful postoperative course and was entirely well when last seen, ten months after the operation.

Pathologic Examination.—The excised sac was composed of amber, friable tissue which in areas was dark red and lobulated. On section, gelatinous and hemorrhagic material exuded from the surface. In many features the tissue resembled degenerated thyroid gland. Microscopically it had the appearance of a degenerated fibromyxoma or a fibromyxosarcoma with much subacute and chronic granulation tissue (fig. 3).

COMMENT

Three cases are reported of malignant tumors involving the sacs of indirect inguinal hernias. All the patients were operated on with a diagnosis of incarcerated hernia, and the presence of a malignant neoplasm was not suspected until histologic study of the excised sac tissues was made. The tumors differed in each case, and all were rather bizarre and difficult of positive histologic identification. In the first case described the patient was an infant, and the tumor was an embryonic type of growth, a mesothelioma of the peritoneum, which was diffuse and involved the subserosal layers of the peritoneum and the pleural diaphragm. It probably originated in multiple centers. Strangely, the only portion of parietal peritoneum involved was the excised portion of the hernial sac. It is likely that ascites resulted from the presence of the tumor, and the appearance of the hernia was due to distention of the sac by ascitic fluid.

The data concerning the other 2 cases are less complete. In the second there was a rather diffuse tumor which involved the abdominal as well as the hernial portions of the wall. It was probably liposarcomatous, although the possibility of its being a metastatic epithelial growth could not be positively ruled out. In the third case a fibromyxosarcoma of the tissues of the hernial sac was present; it was of decidedly lower malignancy and was apparently confined to the tissues of the sac. The patient was well and free from recurrences when last heard from, ten months after the operation.

CONGENITAL ABSENCE OF THE SACRUM

I. M. ZELIGS, M.D.
IOWA CITY

INCIDENCE

Congenital absence of the sacrum, either partial or complete, is of relatively infrequent occurrence. Hamsa,[1] in 1935, could find but 18 cases in the literature. In his review of the subject he included a synopsis of each of these cases and added 2 others. Girard,[2] in the same year, found 7 other cases in the literature and reported 1 of his own. Since that time 11 additional cases have been reported.[3]

ETIOLOGY

There are several etiologic theories. Friedel [4] stated the belief that caudal suppression results from minute embryonal trauma. Wertheim [5] expressed belief in a late prenatal origin. Hamsa [1] stated that the hypothesis of Feller and Sternberg [6] is most plausible, "which explains the sacral variations and peripheral manifestations on the basis of a varying degree of defects in the posterior portions of the metameric segments,

From the Department of Orthopedic Surgery (service of Dr. Arthur Steindler), State University of Iowa Hospitals.

1. Hamsa, W. R.: Congenital Absence of the Sacrum, Arch. Surg. **30**:657 (April) 1935.

2. Girard, P. M.: Congenital Absence of the Sacrum, J. Bone & Joint Surg. **17**:1062 (Oct.) 1935.

3. *(a)* Araújo, A.: Distrofia cruro-vesico-glutea por agenesia total do sacorococcyx, Arq. brasil. de cir. e ortop. **4**:43 (Sept.-Dec.) 1936. *(b)* Barros Lima: A proposito de um caso de malformação do sacro, ibid. **3**:195 (Dec.) 1935. *(c)* Diaz Lira, E.: Agenesia sacrococcigea, Rev. ortop. y traumatol. **7**:231 (Jan.) 1938. *(d)* Guilleminet, M.: Agénésie subtotale du sacrum et du coccyx, Lyon chir. **35**:369 (May-June) 1938. *(e)* Hilgenreiner, H.: Ein Fall von Anchypodie, Ztschr. f. Orthop. **66**:224, 1937. *(f)* Kienböck, R., and Zimmer, A.: Angeborener partieller Kreuz- und Steissbeindefekt, Röntgenpraxis **7**:111 (Feb.) 1935. *(g)* Lamoot and Graux: Agénésie sacro-coccygienne, Echo méd. du Nord **5**:88 (Jan. 19) 1936. *(h)* Müller, J. H.: Ein Fall von Aplasie des Sacrum, Röntgenpraxis **8**:105 (Feb.) 1936. *(i)* Pouzet, F.: Les anomalies de developpement du sacrum, Lyon chir. **35**: 371 (May-June) 1938. *(j)* Willemin, F.: Spondyloptose par agénésie de la base sacrée, J. de radiol. et d'électrol. **21**:57 (Feb.) 1937.

4. Friedel, G.: Defekt der Wirbelsäule vom 10. Brustwirbel an abwärts bei einem Neugeborenen, Arch. f. klin. Chir. **93**:944, 1910.

5. Wertheim, C. C.: Vollständiger Mangel des Kreuz- und Steissbeins bei einem Neugeborenen, Monatschr. f. Geburtsh. u. Frauenkr. **9**:127, 1857.

6. Feller, A., and Sternberg, H.: Zur Kenntnis der Fehlbildungen der Wirbelsäule, Virchows Arch. f. path. Anat. **280**:649, 1931.

with additional involvement of the anterior portions in cases with abdominal visceral anomalies." Whatever the true mechanism is, the condition undoubtedly dates back to early embryonal life. As with other manifestations of suppression, as well as with errors of differentiation, the neurologic and the local variations occur simultaneously.

COMPATIBILITY WITH LIFE

This depends usually on the extensiveness of associated anomalies. When visceral malformations are absent and other significant developmental aberrations are minimal, life is usually possible.

CLINICAL FEATURES

The characteristic clinical features are, in part, manifestations of the vertebral defect and, in part, manifestations of the neurologic lesion which almost inevitably accompanies it. Examples of the former are: (1) a flattening replacing the normal convexity of the sacrococcygeal region; (2) flattening of the buttocks; (3) shortening of the intergluteal fold, and (4) absence of the bony concavity of the sacrococcyx normally palpable on rectal examination.

The neurologic features result from involvement of the sacral or the lumbosacral plexus and include to a varying degree: (1) incontinence of urine and feces; (2) atrophy and diminished muscle power of the lower extremities, and (3) associated deformities of the feet.

The atrophy is frequently more marked below than above the knee, as was pointed out by Hilgenreiner,[3e] with a resultant cone-shaped appearance to the extremities. Such a condition is often referred to as the "sirene" or "mermaid" type. The patient may or may not have adequate muscle power to stand or walk. This frequently depends on the associated congenital deformities. Such deformities are frequent and include:

1. Deformities of the feet. These are present in the great majority of cases. The deformity usually takes the form of paralytic clubfeet. In a number of cases there are equinovarus of one foot and calcaneovalgus of the other. Other deformities have been reported. In the case of Barros Lima [3b] there were marked external rotation deformities of both feet. The patient of de Araújo [3a] showed bilateral ankle valgus, rocker-bottom feet and congenitally short metatarsal bones.

2. Anomalies of other parts of the vertebral portion of the spine. These are also frequently associated conditions. They include suppression of more cranial segments (Friedel,[4] White [7]); hemivertebrae (de Araújo [3a]); failure of development of neural arches (Hilgen-

7. White, C.: A Fetus with Congenital Absence of the Sacrum, Proc. Roy. Soc. Med. **4**:279, 1911.

reiner [3e]); sacralized and wedged fifth lumbar vertebra (Kienböck and Zimmer [3f]), and congenital aplasia of articular apophyses (Diaz Lira [3c]).

3. Dislocation of the hips. This condition has been reported in a number of cases.[8] It is attributed to the decreased transverse diameter of the pelvis and the almost vertical iliac wings.

Among the deformities occurring less frequently are: congenital scoliosis (as in 1 case reported here), genu recurvatum,[8] congenital subluxation of the knee [3e]; congenital synostosis of several ribs, and certain urogenital malformations, such as urethrovaginal fistula or anal atresia (Feller and Sternberg [6]).

Sensory disturbances in the distribution of the lumbar or the lumbosacral plexus occur but do not necessarily parallel the motor changes either in distribution or in severity. In many cases sensory involvement could not be accurately determined owing to the age of the patient.

CLASSIFICATION

Foix and Hillemand [9] have classified the condition according to the extensiveness of involvement of the lower portion of the cord and the vertebral segments into four types:

1. Complete sacrococcygeal agenesis. The sphincters, buttocks and lower extremities are almost always involved. The ilia are frequently approximated posteriorly, with a decrease in the transverse diameter of the pelvic ring. Associated congenital malformations are common and, if extensive, may be incompatible with life. Roentgen examination reveals complete absence of the sacrococcyx.

2. Subtotal sacrococcygeal agenesis. The coccyx and the last few sacral segments are absent. The sphincters, buttocks and lower extremities are involved, and the pelvis is somewhat narrowed. It was for this type that Foix and Hillemand,[9] considering the clinical picture to be a syndrome, introduced the term "dystrophie cruro-vesico-fessiere." The associated congenital conditions are usually less extensive than with type 1.

3. Absence of the lateral half of the sacrum. In this group there are modifications or absence of the sacral wings and univertebral or multivertebral hemiatrophy. There are usually no associated symptoms.

4. Total or partial agenesis of the coccyx. There are usually no symptoms, and the diagnosis is usually made roentgenologically.

The great majority of anomalies reported are of the first two types. The incidence of types 3 and 4 cannot well be determined, since these types are usually asymptomatic and are rarely reported. There are, however, several reports.[10]

---

8. Hamsa.[1] Hilgenreiner.[3e]

9. Foix, C., and Hillemand, P.: Dystrophie cruro-vésico-fessière par agénésie sacro-coccygienne, Rev. neurol. **31**:450 (Nov.) 1924.

(Footnotes continued on next page)

REPORT OF CASES

The files of this clinic reveal 4 cases on record.

CASE 1.—V. F., a girl aged 5 years, has been followed at the clinic since the age of 9 months. She was the sixth child and was born of breech delivery with difficult labor. One sibling, the third child, died of a meningocele. The mother's sister and the father's brother each have a child with congenital clubfeet. The patient has had complete urinary and rectal incontinence since birth.

Physical examination revealed the head, neck, thorax and upper extremities to be normal. The lower extremities were underdeveloped, with moderate atrophy of the calves and, to a lesser extent, of the thighs. There were absence of the

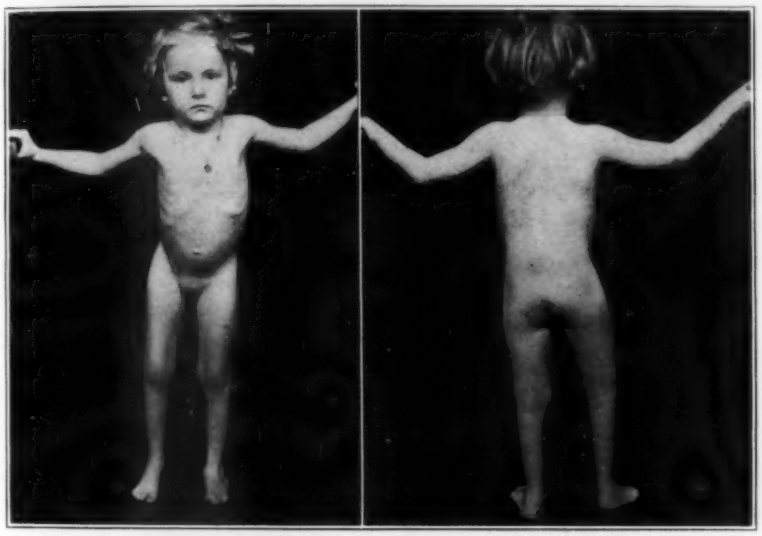


Fig. 1 (case 1).—Flattening of the buttocks, shortening of the intergluteal fold, moderate atrophy of the calves and, to a lesser extent, of the thighs, and mild congenital scoliosis. The clubfeet have been almost fully corrected.

roundness of the sacrococcygeal region and flattening of the buttocks with shortening of the intergluteal fold (fig. 1). There was a suggestion of cone-shaped tapering of both lower extremities. The muscle power of these extremities was moderately impaired, but the child could stand and walk fairly well without support. There were bilateral clubfeet, equinovarus on the right and calcaneovalgus on the left. There was partial paralysis of the long extensor muscle of the great toe on the right and of the common extensor muscle of the toes on the left. Sensation was intact. The deep reflexes of the lower extremities were normal. There was a mild "upper right dorsal, lower left dorsal and right lumbar" scoliosis (fig. 3). There was moderate diastasis recti, with slight abdominal

10. Froelich: Absence congénitale d'une moitié du sacrum, Rev. méd. de l'est **37**:308, 1905. Léri, A.; Pettidi and Cottenot: Anomalie du sacrum, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:1173 (July 18) 1924.

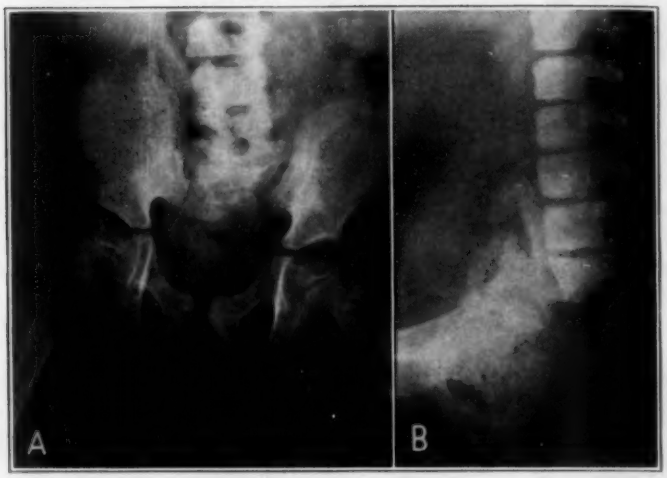


Fig. 2 (case 1).— *A*, malformation of the second sacral vertebral body, with deviation to the right and absence of the sacrococcyx below this level. *B*, lateral view, showing absence of the sacrococcyx below the level of the second sacral vertebra.

Fig. 3 (case 1).—Hemivertebrae in the dorsal part of the spine, with congenital scoliosis, spina bifida in the lower dorsal region, six lumbar vertebrae and partial synostosis of the first and second ribs posteriorly on the right (*A*).

protrusion. Rectal examination revealed poor sphincter tone. Soft tissue was encountered instead of the normal bony concavity of the lower part of the sacrococcyx. There was a congenital malformation of the urethra which opened into the vagina, being separated from the latter by only a thin septum. Partial anal atresia had been noted during infancy and had yielded satisfactorily to digital dilations.

Roentgen examination revealed the following abnormalities:

1. Clubfeet and scoliosis, as described.

2. Congenital partial synostosis of the first and second ribs posteriorly on the right (*A*, fig. 3).

3. Congenital malformation with hemivertebrae from the third to the tenth dorsal vertebra, with spina bifida occulta in the lower part of the dorsal region (fig. 3).

4. Six lumbar vertebrae (fig. 3).

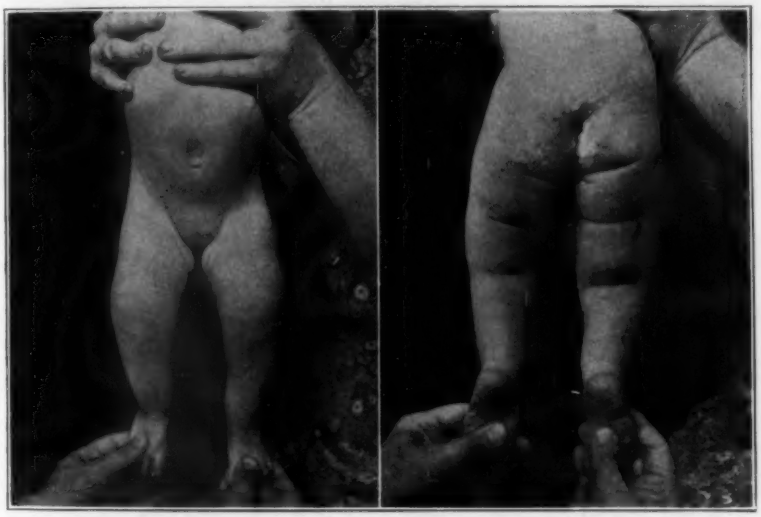


Fig. 4 (case 2).—Absence of the roundness of the sacrococcygeal region, some shortening of the intergluteal fold, tapering of the lower extremities and excoriations and pustules of the buttocks. The clubfeet have been almost fully corrected.

5. Malformation of the second sacral vertebral body with deviation to the right (fig. 2 *A*).

6. Absence of the sacrococcyx below this level (figs. 2 *A* and *B*).

Treatment was designed toward correction of the clubfeet by the usual orthopedic measures. A Brockman operation was necessary on the right side, from which the patient is now convalescing. The child has been equipped with a brace for the left foot. In the past few years she has had several attacks of pyelitis and suppurative otitis media and a number of fecal impactions, from which she has made, however, uneventful recoveries.

Case 2.—R. M. S., a girl aged 7 months, entered the clinic at the age of 4 months for clubfeet. She was the fourth child. She was delivered at home at

full term and weighed 12 pounds (5,443 Gm.) at birth. Labor was difficult and lasted nine hours. Except for pneumonia at the age of 3 months, the child's health had been good. The mother stated that she had had difficulty in keeping the baby clean and dry. The parents and three siblings were in good health. There was no family history of similar deformities.

Physical examination revealed the head, neck, thorax and upper extremities to be normal. The lower extremities presented a peculiar cone-shaped appearance, the thighs being large and flabby and the legs from the knees downward progressively thinner. There were absence of the roundness of the sacrococcygeal region and some shortening of the intergluteal fold (fig. 4). As the child was held in a sitting position, there was a soft tissue bulge dorsally at the lumbosacral region. Bilateral talipes equinovarus was noted. As far as could be determined at this age, muscle power and sensation in the lower extremities were unimpaired. There were moderate flexion contractures of the hips and knees.

Fig. 5 (case 2).—Three lumbar vertebrae and a rudimentary fourth, with spina bifida occulta articulating laterally with the iliac wings. The fifth lumbar vertebra is absent, as are the sacrum and the coccyx. The lateral view shows absence of the fifth lumbar vertebra, the sacrum and the coccyx.

Rectal examination revealed poor sphincter tone. Soft tissue was encountered instead of the bony concavity of the sacrococcyx. Incontinence of urine and feces was noted as the child cried. There were extensive excoriations and numerous pustules over the vulva and buttocks, due to the urine, which seemed to flow almost constantly (fig. 4). There was a moderate degree of forward angulation of the pelvis.

Roentgen examination revealed the following abnormalities (fig. 5):

1. Clubfeet as described.

2. Three lumbar vertebrae and a rudimentary fourth, with a spina bifida occulta articulating laterally with the iliac wings.

3. Absence of the fifth lumbar vertebra.

4. Complete absence of the sacrum and coccyx.

Treatment was designed toward correction of the clubfeet in the usual manner. These yielded satisfactorily to corrective casts during a period of three months. The child is now at the hospital for treatment of the contractures of the hip and knee.

A brief résumé of cases 3 and 4, previously reported by Hamsa,[1] with a more recent follow-up, is given.

CASE 3.—S. J., a girl aged 14 months, was born at full term of a 20 year old primipara by normal delivery. Deformities of the spine and legs and shortening of the thighs and torso were noted at birth. There was incontinence of urine and feces. The patient's family history was unimportant. The abnormalities included: a short torso, with lumbar kyphosis; dislocation of both hips; clubfeet; flattening of the buttocks, and some atrophy of the thighs. Roentgen examination further revealed: spina bifida occulta of the upper part of the dorsal region; partial or complete fusion of the ribs; a right lumbar rib; three rudimentary lumbar vertebrae, and absence of the sacrum and coccyx, with the iliac wings almost touching posteriorly. There was also loss of osseous continuity between the third lumbar vertebra and the pelvis. At the age of 2 years the child could sit alone and could stand by bracing her knees against each other. Operative correction of the feet was advised at that time, but the patient did not return for treatment. A recent follow-up revealed that at the age of 8 years the patient showed little change. She weighed 30 pounds (13.6 Kg.), had grown very little and still had no sphincter control. She required a daily enema. She could not walk. No reduction of the dislocated hips was done owing to the malformation of the pelvis.

CASE 4.—R. S., a 9 year old boy, of normal birth, had four lumbar vertebrae and absence of the sacrum and coccyx, with the iliac wings in close apposition posteriorly. There were congenitally dislocated hips and deformities of the lower extremities. The latter consisted of bilateral ankle valgus and 50 degrees of recurvatum of the left knee, with only a few degrees of flexion from complete extension possible in the right knee. There was atrophy of the thighs and buttocks, with a short intergluteal fold. Urinary and fecal incontinence were complete, but the latter gradually improved. After operative alinement of the lower extremities, the patient could walk with braces and crutches. When he was last seen, two years prior to this report, at the age of 19, there was still considerable weakness of the lower extremities, necessitating crutches. There was good control of the bowels but not of the bladder. Both ankles were in valgus. The patient was getting about, however, and was attending business college. No further indication for treatment was made at that time.

COMMENT

The characteristic features of partial or complete sacrococcygeal agenesis are well illustrated in the cases presented and resemble closely in their essential features the cases in the literature. Case 1 fits well into group II of Foix and Hillemand's classification, while the other 3 cases are just as typical of group I. The loss of sphincter control is one of the most disturbing features. Spontaneous improvement, especially of the fecal incontinence, occurs, however, as is shown by case 4. Some patients have fecal but not urinary incontinence (Barros Lima [3b]); others have the reverse (de Araújo [3a]).

The case of Diaz Lira [3c] is of interest in that it is the only instance in the literature in which neurolysis of the cauda equina was attempted for the condition. The indication for surgical treatment here, however, followed the development of rectal incontinence and diminution in the deep reflexes of the lower extremities at the age of 3 years. On the basis of these manifestations the cauda equina was explored and scar and fatty tissue were removed from the conglomerate sacral roots, with progressive improvement several days after operation, not only in the rectal incontinence which had recently developed but in the urinary incontinence which had been present since birth. (The patient was a 9 year old boy with conical lower extremities, clubfeet, incontinence of urine and constipation present since birth. There was agenesis of the coccyx and of the last three sacral segments.)

Of interest also is Willemin's [3j] case of a 21 year old man with complete agenesis of the sacral base corresponding to the first sacral segment. The rest of the sacrococcyx was normal. A marked spondylolisthesis of the fifth lumbar vertebra developed. There were no sphincter disturbances, and the lower extremities were normal. This case was the only one of this type of agenesis encountered and does not fit into the classification of Foix and Hillemand.[9] Willemin, however, in his article, stated that he knows of 4 other cases of similar involvement.

The treatment is ordinarily limited to correction of the deformities of the lower extremities by the usual orthopedic measures, as is shown in 3 of the cases reported.

SUMMARY

To the 39 cases of partial or complete sacrococcygeal agenesis reported in the literature 2 cases are added, with a review and a recent follow-up study of 2 other cases previously reported from this clinic. The characteristic features and classification of the condition are reviewed. The former include varying degrees of urinary and rectal incontinence, flattening of the buttocks and atrophy and weakness of the lower extremities, with absence of the coccyx and of all or part of the sacrum. Treatment consists of the usual orthopedic measures designed to correct the existing deformities of the lower extremities when enough muscle power is present to enable the patient to assume the erect position.

FEMORAL HERNIAS

A STUDY OF TWO HUNDRED AND THIRTY-EIGHT HERNIAS AND TWO HUNDRED AND TWENTY-SIX REPAIRS

HAROLD J. SHELLEY, M.D.
FORT WORTH, TEXAS

Included in this study were 238 femoral hernias.[1] They comprised 5.35 per cent of the total group of all types of hernias seen in the wards in the period covered by this study. Among these, femoral hernias not previously repaired numbered 222, of which 210 were repaired by operation. Of these, 140 were examined postoperatively for nine months or longer or until a recurrence was discovered. Only 5 recurrences developed, giving a recurrence rate of 3.6 per cent.

The remaining 16 femoral hernias were recurrent, following a previous repair. All 16 were operated on. Thirteen were followed for nine months or longer, and 2 recurrences were discovered, a recurrence rate of 15.4 per cent.

The period covered by this study was from 1916 to 1935 inclusive. All femoral hernias in patients admitted to the wards at St. Luke's Hospital, New York, from 1926 to 1935 and all hernias of this type repaired in the ten year period (1916 to 1925) in patients who returned for follow-up examinations over periods of nine months or longer, or until a recurrence was discovered, were included in this study.

ETIOLOGIC FACTORS

Age at Which Hernia Was First Noted.—Primary Hernias: The average age of the patients when these 222 femoral hernias were first noted was 39.6 years.

In the first two decades of life, only 3.2 and 4.1 per cent respectively of these hernias were first noted. This figure was increased to 19.8 per cent for the third decade. The incidence for the fourth, of 25.2 per cent, was the greatest for any decade, although it was only slightly above

---

From the Surgical Services of St. Luke's Hospital.

1. All references to types of hernias other than femoral are from the following papers published by me: Incomplete Indirect Inguinal Hernias: A Study of 2,462 Hernias and 2,337 Repairs, Arch. Surg. **41**:747-771 (Sept.) 1940; Complete Indirect Inguinal Hernias: A Study of 305 Hernias and Repairs, South. Surgeon **9**:257-268 (April) 1940; Direct Inguinal Hernias: A Study of Six Hundred and Five Hernias and Five Hundred and Sixty-Five Repairs, Arch. Surg. **41**:857 (Oct.) 1940; Ventral Hernias: A Study of 550 Hernias and 458 Repairs, South Surgeon **9**:617-656 (Sept.) 1940.

TABLE 1.—*Age at Which Hernia Was First Noted*

Age Group, Years	Total Hernias	Per Cent of Entire Group		Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences	
0 to 10	7	3.2	7.3	3	1	33.3	12.5
10 to 20	9	4.1		5	0	0.0	
20 to 30	44	19.8	45.0	27	1	3.7	1.6
30 to 40	56	25.2		36	0	0.0	
40 to 50	54	24.3	39.2	44	2	4.5	4.9
50 to 60	33	14.9		19	1	5.3	
60 to 70	11	5.0	8.7	5	0	0.0	0.0
70 to 80	7	3.2		1	0	0.0	
80 to 90	1	0.5		0	..	..	
Totals.....	222	100.0	100.0	140	5	3.6	3.6

The average age at which the femoral hernias were first noted was 39.6 years.

TABLE 2.—*Age at Time of Admission or Operation (Femoral Hernias)*

Age Group, Years	Total Hernias	Per Cent of Entire Group		Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences	
0 to 10	3	1.3	4.4	1	0	0.0	0.0
10 to 20	7	3.1		6	0	0.0	
20 to 30	33	14.8	34.1	18	1	5.5	4.2
30 to 40	43	19.3		30	1	3.3	
40 to 50	64	28.8	48.6	46	1	2.1	2.7
50 to 60	44	19.8		29	1	3.4	
60 to 70	17	7.6	12.5	8	1	12.5	10.0
70 to 80	9	4.0		2	0	0.0	
80 to 90	2	0.9		0	..	...	
Totals.....	222	100.0	100.0	140	5	3.6	3.6

The average age of the patients at the time of admission to the hospital or operation for femoral hernias was 43.7 years.

TABLE 3.—*Age at Time of Admission or Operation (Recurrent Femoral Hernias)*

Age Group, Years	Total Hernias	Per Cent of Entire Group		Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences	
20 to 30	1	6.3	25.0	1	0	0.0	25.0
30 to 40	3	18.7		3	1	33.3	
40 to 50	4	25.0	56.2	3	0	0.0	14.3
50 to 60	5	31.2		4	1	25.0	
60 to 70	2	12.5	18.8	1	0	0.0	0.0
70 to 80	1	6.3		1	0	0.0	
Totals.....	16	100.0	100.0	13	2	15.4	15.4

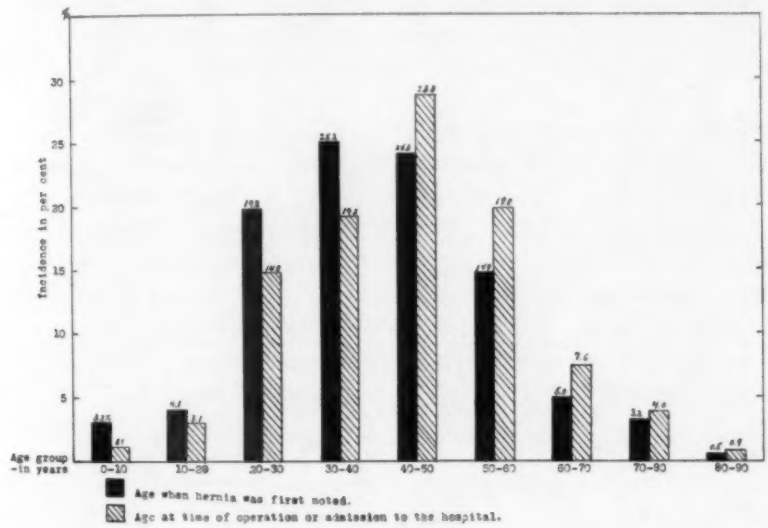
The average age of the patients at the time of admission to the hospital or operation for recurrences of femoral hernias was 49 years.

the figure for the fifth decade, 24.3 per cent. The incidence decreased through the succeeding decades to 0.5 per cent for the ninth decade. The incidence for the time covering the child-bearing period of women's lives, from 20 to 50, was 69.3 per cent of all the hernias.

The incidences for the successive twenty year periods of life were 7.3, 45, 39.2 and 8.7 per cent respectively.

The number of recurrences was too small for the recurrence rates according to the ages at which the hernias were first noted to have any particular significance.

Recurrent Hernias: The average age of the patients when these recurrences were first noted was 41.6 years, 2.1 years less than the



Age incidence.

average age at which the primary repairs were done, 43.7 years. Evidently, fewer of the older patients with recurrences came into the hospital for operation. The age of the youngest patient at the time a recurrence was first noted was 22 years, and that of the oldest, 79 years.

The number of repairs was too small for the recurrence rates according to the patients' ages to have any significance.

Age at Admission or Operation.—Primary Hernias: The average age of the patients with these hernias was 43.7 years.

The age incidences at the time of admission or operation increased from 1.3 per cent for the first decade of life to 28.8 per cent for the fifth. From that period it decreased to 0.9 per cent for the ninth decade. Each decade from the third to the ninth showed 1 recurrence.

The incidences for the successive twenty year periods of life were 4.4, 34.1, 48.6 and 12.5 per cent respectively.

TABLE 4.—*Sex (Femoral Hernias)*

Sex	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences
Male	85	38.3	55	0	0.0
Female	137	61.7	85	5	5.9
Totals	222	100.0	140	5	3.6

TABLE 5.—*Sex (Recurrent Femoral Hernias)*

Sex	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences
Male	3	18.7	1	0	0.0
Female	13	81.3	12	2	16.7
Totals	16	100.0	13	2	15.4

TABLE 6.—*Race (Femoral Hernias)*

Race	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences
White	217	97.7	138	5	3.6
Negro	5	2.3	2	0	0.0
Totals	222	100.0	140	5	3.6

TABLE 7.—*Race (Recurrent Femoral Hernias)*

Race	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences
White	16	100.0	13	2	15.4
Negro	0	0.0	..	..	....
Totals	16	100.0	13	2	15.4

TABLE 8.—*History of Definite Trauma as Etiologic Factor* *

History of Trauma as Cause*	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences
Absent	185	83.3	115	5	4.3
Positive	37	16.7	25	0	0.0
Totals	222	100.0	140	5	3.6

* Undoubtedly all or a major part of these hernias were present before the incidence of the trauma, which served only to call the patients' attention to the presence of the hernia. In this connection it is interesting to note that among the 305 complete (congenital) indirect inguinal hernias studied a history of definite trauma as the etiologic factor in the development of their hernias was given by 26 per cent of the patients who first noted their hernias after the age of 15 years (Shelley, H. J.: Complete Indirect Inguinal Hernias: A Study of 305 Hernias and Repairs, South. Surgeon **9**: 257-268 [April] 1940).

Recurrent Hernias: The youngest patient at the time of operation for recurrent femoral hernias was 26 years, the oldest, 79 years. The average age was 49 years. This was the greatest average age at the time of repair for all types of inguinal and femoral hernias. The incidence of the patients' ages at the time of operation increased from the third to the sixth decades of life and then decreased to the eighth.

Sex.—Primary Hernias: Of the 222 primary femoral hernias studied, 61.7 per cent were found in females and 38.3 per cent in males. All of the 5 recurrences were in female patients. None were discovered in the 55 repairs performed on male patients which were observed post-operatively for nine months or longer.

Recurrent Hernias: The predominance of females among the patients operated on for recurrent femoral hernias, 81.3 per cent, was considerably greater than that found in the case of the original repairs, 61.7 per cent.

Race.—Primary Hernias: Only 5 patients with these hernias were Negroes, 2.3 per cent; 97.7 per cent were white.

Recurrent Hernias: All 16 patients having recurrent femoral hernias were members of the white race.

Trauma.—Primary Hernias: A history of definite trauma as the etiologic factor in the development of these hernias was given by 16.7 per cent of the patients. (See note under table 8.) That child-bearing may be a definite etiologic factor in their development is indicated by their greatly increased incidence during the years in which women bear children (table 1).

No recurrences were found in the 25 followed patients who had given a history of a definite trauma as the etiologic factor in the development of their hernias.

Recurrent Hernias: None of the 16 patients with recurrent hernias gave a history of a definite trauma as the cause of the recurrence.

SYMPTOMS

Pain.—Primary Hernias: As would be expected with hernias having a small opening into the abdominal cavity, about which is a fairly rigid border, a history of pain associated with their hernias was given by a larger proportion of these patients, 70.7 per cent, than was found to be the case with any type of inguinal hernia.

Recurrent Hernias: An even greater proportion of patients, 87.5 per cent, with recurrent femoral hernias gave a history of pain associated with the presence of their hernias.

Duration.—Primary Hernias: Somewhat less than half (44.1 per cent) of these hernias were repaired within the first year after they were first noted. The recurrence rate, 1.6 per cent, when they were repaired within the first year was less than half that for the entire group. More

than two thirds, 68 per cent, were repaired within five years, and 82.9 per cent were repaired within ten years, of the time when they were first noted.

TABLE 9.—*History of Pain Associated with Femoral Hernia*

History of Pain	Total Hernias	Per Cent of Entire Group
Absent	65	29.3
Positive	157	70.7
Totals	222	100.0

TABLE 10.—*History of Pain Associated with Recurrent Femoral Hernia*

History of Pain	Total Hernias	Per Cent of Entire Group
Absent	2	12.5
Positive	14	87.5
Totals	16	100.0

TABLE 11.—*Duration (Time Hernia Was First Noted to Time of Admission or Operation)*

Duration	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences
To 1 week	26	11.7	16	0	0.0
To 1 month	39	17.6	21	0	0.0
First 6 months	74	33.3	44	0	0.0
Second 6 months	24	10.8	19	1	5.3
To 1 year	98	44.1	63	1	1.6
0 to 5 years	151	68.0	100	3	3.0
5 to 10 years	33	14.9	23	0	0.0
0 to 10 years	184	82.9	123	3	2.4
10 to 20 years	27	12.2	13	0	0.0
20 to 30 years	9	4.1	3	2	66.7
30 to 40 years	1	0.5	1	0	0.0
40 to 50 years	1	0.5	0	..	...
Totals	222	100.0	140	5	3.6

The average duration (from the time when these hernias were first noted to the time of admission or operation) for femoral hernias was 4.1 years.

The average duration (from the time these hernias were first noted to the time of the patient's admission to the hospital) was four and one-tenth years. This was a greater duration than was observed with any type of inguinal hernia with the exception of complete indirect inguinal hernia.

Recurrent Hernias: For these hernias the average duration was seven and four-tenths years, the greatest for any type of inguinal or femoral hernias.

PHYSICAL FINDINGS

Size.—Primary Hernias: The sacs of over half (51.8 per cent) of these femoral hernias were not more than 2½ inches (6.2 cm.) in

TABLE 12.—*Size*

Size of Hernia *	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences	Operative Deaths (1926-1935)†	Per Cent Mortality
Size I	115	51.8	70	3	4.3	3 in 74 operations	4.1
Size II	89	40.1	57	2	3.5	3 in 58 operations	5.2
Size III	18	8.1	13	0	0.0	1 in 12 operations	8.3
Totals	222	100.0	140	5	3.6	7 in 144 operations	4.9

* Size I: Hernias in which the greatest diameter of the sac was not more than 2½ inches (6.2 cm.).
Size II: Hernias in which the greatest diameter of the sac was more than 2½ inches and not more than 3½ inches (8.8 cm.).
Size III: Hernias in which the greatest diameter of the sac was more than 3½ inches (8.8 cm.).
† All 7 deaths were of patients entering the hospital with strangulated hernias.

TABLE 13.—*Unilateral and Bilateral Hernias* *

	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences
		(A)			
Unilateral, right	136	61.3	89	3	3.3
Unilateral, left	63	28.4	39	2	5.1
Total unilateral	198	89.2	128	5	3.9
Total bilateral	24	10.8	12	0	0.0
Total right	148	66.7	95	3	3.2
Total left	74	33.3	45	2	4.4
Totals	222	100.0	140	5	3.6
		(B)			
Bilateral hernias, each repaired separately					20
Bilateral hernias, both repaired at one sitting					4
No recurrences.					

* Each hernia was counted individually. Each femoral hernia was considered as one of two bilateral hernias when there was or had been a femoral hernia on the opposite side.

diameter. Slightly less, 40.1 per cent, had sacs over 2½ inches and less than 3½ inches (8.8 cm.) in diameter, and only 8.1 per cent had sacs more than 3½ inches in diameter.

The number of recurrences was too small to give definite indications as to the relation of the size of the sac to the probability of recurrence.

It is interesting to note that the mortality figure increased with increase in size of these hernias, but the number of hernias in each group was possibly too small for these figures to be conclusive.

Recurrent Hernias: The groupings of these hernias according to the size of the sac as outlined was 25 per cent, 37.5 per cent and 37.5 per cent respectively, representing a marked decrease in the smallest size and a correspondingly marked increase in the largest.

Among the repairs of the smaller hernias were no operative deaths or recurrences. After repairs of the largest hernias, the mortality rate was 25 per cent and the recurrence rate 20 per cent.

Unilateral and Bilateral Hernias.—Primary Hernias: The incidence of bilateral femoral hernias, 10.8 per cent, was only one third that of incomplete indirect inguinal hernias, 32.6 per cent. The incidence of

TABLE 14.—*Incarceration and Strangulation (Femoral Hernias)*

Incarcerated or Strangulated	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences	Operative Deaths (1926-1935)	Per Cent Mortality
Neither	102	45.9	66	3	4.5	0 in 52 operations	0.0
Incarcerated *	94	42.4	59	2	3.4	3 in 74 operations*	4.1
Strangulated *	37	16.7	20	0	0.0	7 in 27 operations*	25.9
Totals	222	100.0	140	5	3.6	7 in 144 operations*	4.9

* Strangulation developed in 11 old incarcerated hernias. Strangulation was present in all 7 patients who died postoperatively. In 3 of these the hernia had been incarcerated previous to the development of the strangulation.

femoral hernias on the right side, 66.7 per cent, was greater than the incidence of incomplete indirect inguinal hernias on the right side, 55 per cent.

The probability of recurrence following repair contrasting unilateral with bilateral and right with left femoral hernias cannot be given from this study, as not a sufficiently large number were studied in each group for the recurrence figures to have any significance.

Recurrent Hernias: In 12.5 per cent of the patients with recurrent femoral hernias there was a femoral hernia on the opposite side at the same time, or there had previously been one. This is essentially the same as the incidence of bilateral primary femoral hernias, 10.8 per cent.

In such a small number of repairs, the recurrence rates cannot be taken as significant as to the probability of recurrence when these hernias are unilateral or bilateral.

Incarceration and Strangulation.—Primary Hernias: The incidences of incarceration and strangulation were considerably increased in the femoral hernias as compared to incomplete indirect inguinal hernias. This is the natural expectation because of the small opening into the

abdominal cavity with a relatively rigid circumference. Of the 222 femoral hernias, 42.4 per cent were incarcerated, as contrasted to only 6 per cent in the incomplete indirect inguinal hernias. The figures were 16.7 per cent and 1.8 per cent respectively for strangulation.

So far as the respective incidence rates of the small number of recurrences would indicate, incarceration and strangulation bore no relation to the probability of recurrence following repairs of femoral hernias.

However, these two conditions had a direct bearing on the mortality following operative repair. In the presence of incarceration of femoral hernias the mortality rate was 4.1 per cent, but all the deaths making up this figure were associated with incarcerated hernias which had later become strangulated. For strangulated femoral hernias the mortality rate in 27 operations was 25.9 per cent. There were no deaths associated with the 52 operations on femoral hernias which were neither

TABLE 15.—*Incarceration and Strangulation (Recurrent Femoral Hernias)*

Incarcerated or Strangulated	Total Hernias	Per Cent of Entire Group	Number Followed Postoperatively	Number of Recurrences	Per Cent Recurrences	Operative Deaths (1926-1935)	Per Cent Mortality
Neither	5	31.2	5	1	20.0	0 in 5 operations	0.0
Incarcerated *	10	62.5	8	1	12.5	1 in 10 operations†	10.0
Strangulated *	3	18.7	1	0	0.0	1 in 3 operations†	33.3
Totals	16	100.0	13	2	15.4	1 in 16 operations†	6.3

* Strangulation developed in 2 old incarcerated hernias.
† The patient who died had strangulation in an old incarcerated hernia.

incarcerated nor strangulated. (Mortality rates were calculated only on the repairs performed between 1926 and 1935.)

Recurrent Hernias: Larger percentages of these hernias were incarcerated, 62.5 per cent, and strangulated, 18.7 per cent, than was the case with femoral hernias which were not recurrent, 42.4 and 16.7 per cent respectively.

The relation of incarceration and strangulation to the incidence of recurrence and mortality was essentially the same as with the original femoral repairs, with the exception of an increased percentage in each instance. The only patient who died after operation had a strangulated hernia, but this hernia was incarcerated before the development of strangulation.

RESULTS ACCORDING TO TYPE OF OPERATIONS

Primary Hernias.—The variations in the technic of repairing these hernias were considered too slight to warrant tabulating the figures as to results according to the different types of repair. The number of recurrences was so small that the figures would have had no value in deter-

mining the comparative success of these operations. All hernias were repaired from below Poupart's ligament. This structure was sutured to the pectineus fascia with ordinary sutures, mattress sutures or, in a few instances, a purse string suture. Silk was used in only a few repairs.

Recurrent Hernias.—All but 1 of these recurrent hernias were repaired with catgut suture material alone. The recurrence rate for these repairs was 16.7 per cent, and in 20 per cent of these operative wounds infection developed.

TABLE 16.—*Postoperative Respiratory Complications*

Complication	Total Operations	Per Cent of Entire Group	Average Postoperative Stay in Hospital, Days	Number Followed Postoperatively	Number of Recurrences	Per Cent of Recurrences	Per Cent of Infected Wounds	Deaths	Per Cent Mortality
1. Acute bronchitis	6	2.9	16.5	3	0	0.0	0.0	0	0.0
2. Lobar pneumonia	2	0.9	16.0	1	0	0.0	50.0	1	50.0
3. Bronchopneumonia	1	0.48	4.0	0	..	...	0.0	1	100.0
4. Acute tonsillitis	1	0.48	15.0	1	0	0.0	0.0	0	0.0
Totals	10	4.8		5	0	0.0	10.0	2	20.0

TABLE 17.—*Postoperative Circulatory Complications*

Complication	Total Operations	Per Cent of Entire Group	Average Postoperative Stay in Hospital, Days	Number Followed Postoperatively	Number of Recurrences	Per Cent of Recurrences	Per Cent of Infected Wounds	Deaths	Per Cent Mortality
1. Hematomas	3	1.4	14.6	3	0	0.0	0.0	0	0.0
2. Deep phlebitis	3	1.4	24.0	3	0	0.0	0.0	0	0.0
Totals	6	2.8		6	0	0.0	0.0	0	0.0

Only one repair was performed with a fascial suture from the fascia lata. This wound healed without infection, and no recurrence had developed when the patient was last examined, two years after the operation.

POSTOPERATIVE COMPLICATIONS

Primary Hernias.—The incidence of postoperative complications was 50 per cent greater after repair of femoral hernias (15.6 per cent) than after repair of incomplete indirect inguinal hernias (10.4 per cent). The reason for this was not apparent. The major complications appeared in much the same order, but deep phlebitis, which followed only 0.3 per cent of the inguinal repairs, was found after 1.4 per cent of the femoral repairs.

The incidence of respiratory complications, 4.8 per cent, was only slightly more than half that found after repairs of incomplete indirect inguinal hernias, 7.3 per cent.

TABLE 18.—*Miscellaneous Postoperative Complications*

Complication	Total Operations	Per Cent of Entire Group	Average Postoperative Stay in Hospital, Days	Number Followed Postoperatively	Number of Recurrences	Per Cent of Recurrences	Per Cent of Infected Wounds	Deaths	Per Cent Mortality
1. Wound infection.....	11	5.2	17.8	8	0	0.0	...	0	0.0
2. Acute bronchitis.....	6	2.9	16.5	3	0	0.0	0.0	0	0.0
3. Hematomas.....	3	1.4	14.6	3	0	0.0	0.0	0	0.0
4. Deep phlebitis.....	3	1.4	24.0	3	0	0.0	0.0	0	0.0
5. Lobar pneumonia.....	2	0.9	16.0	1	0	0.0	50.0	1	50.0
6. Bronchopneumonia...	1	0.48	4.0	0	..	...	0.0	1	100.0
7. Acute tonsillitis.....	1	0.48	15.0	1	0	0.0	0.0	0	0.0
8. Cystitis.....	1	0.48	19.0	1	0	0.0	0.0	0	0.0
9. Pyelitis.....	1	0.48	60.0	0	..	...	0.0	0	0.0
10. Acute appendicitis....	1	0.48	21.0	1	0	0.0	0.0	0	0.0
11. Acute cholecystitis....	1	0.48	29.0	1	0	0.0	0.0	0	0.0
12. Paralytic ileus *.....	1	0.48	1.0	0	..	...	0.0	1	100.0
13. Bacillus welchii peritonitis *.....	1	0.48	2.5	0	..	...	100.0	1	100.0
Totals.....	33	15.6		22	0	0.0	6.1	4†	12.1

* These complications both developed in patients who had had strangulated hernias.
† Three patients died as a result of the toxemia from the preoperative intestinal obstruction.

TABLE 19.—*Deaths (Femoral Hernias, 1926-1935)*

Operative Deaths *	Patient's Age	Hernia Strangulated	Duration of Strangulation	Resection Required	Time of Death (Postoperative)	Cause of Death
1	44	Yes	18 hours	No †	2½ days	B. welchii wound infection and peritonitis
2	52	Yes	1 week	No	Immediately	Toxemia from long-standing strangulation and shock
3	69	Yes	3 days	No	3 days	Lobar pneumonia; coronary fibrosis; myocardial sclerosis; pyelonephritis
4	69	Yes	3 days	No	4 days	Bronchopneumonia; arteriosclerotic heart disease
5	70	Yes	4 days	No	12 hours	Toxemia from long-standing strangulation; shock
6	87	Yes	6 days	Yes	24 hours	Paralytic ileus
7	68	Yes	4 days	‡	10 hours	Toxemia; intestinal obstruction

* Seven deaths in 144 operations, giving an operative mortality of 4.9 per cent.
† Small area of gangrene in a Richter type hernia. The area of gangrene was inverted.
‡ Enterostomy in the strangulated loop. No repair of the hernia was done.

TABLE 20.—*Deaths (Recurrent Femoral Hernias, 1926-1935)*

Operative Deaths *	Patient's Age	Hernia Strangulated	Duration of Strangulation	Resection Required	Time of Death	Cause of Death
1	79	Yes	6 hours	No	2 hours after operation	Ileus; shock

* One death in 16 operations, giving an operative mortality of 6.3 per cent.

The incidences of circulatory complications were the same following repairs of femoral and of incomplete indirect inguinal hernias, 2.8 per cent.

Recurrent Hernias.—The incidence of postoperative complications was greater after repair of recurrences than after the original operations. Complications developed after 18.8 per cent of these operations, as compared to 15.6 per cent of original femoral repairs. Respiratory complications followed 6.3 per cent of the repairs of recurrences, as compared with 4.8 per cent, and circulatory complications after 6.3 per cent, as compared with 2.8 per cent.

OPERATIVE MORTALITY

Primary Hernias.—The operative mortality of 4.9 per cent (as compared to 0.52 per cent in incomplete indirect inguinal hernias) was due

TABLE 21.—*Follow-Up Data (Femoral Hernias)*

Total number of hernias studied	222
Total with no operation	12
Total operations	210
Deaths (all operative)	7
Total with no follow-up examination	34
Total with follow-up under 9 months (no recurrences)	30
Total with follow-up 9 months and over (including recurrences)	140
Average follow-up time (all cases followed 9 months and over)	24.9 months
Total recurrences	5
Average postoperative time recurrences were first noted	26.8 months
Percentage of recurrences (follow-up 9 months and over)	3.6
Total number of operations examined postoperatively	170
Average follow-up time (all followed cases)	21.2 months
Total recurrences	5
Percentage of recurrences (all followed cases)	2.9

to 7 deaths, all following repairs of strangulated hernias. The various facts about these 7 deaths are given in table 19.

Recurrent Hernias.—One death followed repair of 16 recurrent femoral hernias, a mortality rate of 6.3 per cent. The patient, who was 79 years of age, had had a strangulation for six hours. She died two hours after the operation, of ileus, shock and preoperative toxemia and dehydration. No resection was required.

FOLLOW-UP DATA

Primary Hernias.—Of the 222 femoral hernias studied, 12 were not repaired. There were 7 operative deaths, and 34 patients did not return for follow-up examination. One hundred and forty were followed for nine months or longer, for an average period of twenty-four and nine-tenths months. Only 5 recurrences were discovered, an incidence of 3.6 per cent. The average postoperative time at which the recurrences were first noted was twenty-six and eight-tenths months.

A total of 170 were examined in the follow-up clinic. The average follow-up time for all followed repairs was twenty-one and two-tenths months. The recurrence rate calculated on all follow-up examinations was 2.9 per cent.

Recurrent Hernias.—All of the 16 patients with recurrent femoral hernias were operated on. One died postoperatively. Thirteen of the remaining 15 were followed for nine months or longer. The average follow-up period was thirty-three and two-tenths months. Two recurrences were discovered, giving a recurrence rate of 15.4 per cent. The average postoperative time at which these recurrences were discovered was ten months.

Fourteen patients altogether were examined in the follow-up clinic. The average follow-up time was thirty and one-tenth months and the recurrence rate 14.3 per cent.

TABLE 22.—*Follow-Up Data (Recurrent Femoral Hernias)*

Total number of hernias studied	16
Total with no operation	0
Total operations	16
Total operative deaths	1
Total with no follow-up examination	1
Total with follow-up under 9 months (no recurrences)	1
Total with follow-up 9 months and over (including recurrences)	13
Average follow-up time (all cases followed 9 months and over)	32.2 months
Total recurrences	2
Average postoperative time when recurrences were first noted	10.0 months
Percentage of recurrences (follow-up 9 months and over)	15.4
Total number of operations examined postoperatively	14
Average follow-up time (all followed cases)	30.1 months
Total recurrences	2
Percentage of recurrences (all followed cases)	14.3

REPAIR OF FEMORAL HERNIAS

Primary Hernias.—It is probably unnecessary to go into great detail as to the technic of repair of femoral hernias. With careful, intelligent surgical handling, satisfactory results will ensue whatever the method of repair.

The question of the type of incision in the skin and subcutaneous tissues is largely that of the individual preference of the surgeon. In the presence of an inguinal hernia associated with a femoral hernia, naturally an oblique incision should be used. In those few instances in which there is a possibility that the hernia may be inguinal instead of femoral, again the oblique incision should be used.

The necessity of an approach to the neck of the sac from above the inguinal ligament will present in few repairs if the surgeon is familiar with the anatomy of the femoral region and the technic of carrying out the various necessary procedures in that region.

Careful, clean dissection of the structures about the femoral ring is an important point. Location of the interior of the sac without injury to its contents at times will present certain difficulties. This is due to (1) adherence of the contained omentum or intestine to the peritoneal lining of the sac; (2) cystic degeneration of the incarcerated omentum, giving false pockets simulating the interior of the sac, and (3) edema or even cystic degeneration of the fat and other tissues making up the hernial sac itself. This difficulty can usually be avoided by opening the sac near the neck, where these various conditions do not ordinarily present themselves.

When the femoral canal is too small for reduction of the contents of the sac into the abdomen, incision of Gimbernat's ligament will ordinarily suffice to permit the contents to be returned to the abdomen. An occasional rare case will be observed in which additional careful enlargement of the femoral ring by retraction laterally will be necessary.

I believe that when a resection is necessary it should be done through a lower rectus incision. Reduction of the anastomosed loops of intestine through the femoral opening may damage the anastomosis, and the resection itself may well contaminate the operative wound and thereby greatly increase the probability of recurrence of the hernia.

In my experience, reduction of the strangulated loop for a time into the abdominal cavity has frequently demonstrated that a resection was unnecessary, while observation of that same strangulated loop in the operative wound, even though the femoral opening had been apparently adequately enlarged, would indicate that resection was required.

Avoidance of unnecessary resections will reduce markedly the operative mortality associated with strangulated femoral hernias.

As to closure of the femoral canal itself, any of the various technics, if properly applied, will give satisfactory results. My preference in this respect is for mattress sutures reenforced by two or three ordinary sutures. The lateral suture should be applied with the femoral vein carefully retracted laterally. In closure of the wound, all dead spaces should be carefully eliminated. Use of silk throughout for suture and ligature material is a distinct improvement over use of catgut.

With the exception of those cases in which there is an unusually large femoral opening and those in which this opening has been markedly enlarged in order to reduce the contents of the sac, these patients do not require as long a period of time in bed postoperatively as should be insisted on after repairs of inguinal hernias. However, it is even more important that these patients have their position in bed changed frequently immediately after the operation and that active movements be instituted as early as possible and kept up until the patients are permitted out of bed. In this series of repairs the incidence of deep phlebitis was four times that which followed repair of incomplete indirect inguinal hernias.

Recurrent Hernias.—Repair of the majority of recurrent femoral hernias will not differ materially from repair of primary hernias except that dissection of the sac and the various anatomic structures will present added difficulties due to the scar of the previous operation. In most cases a careful repair, preferably with mattress sutures, will give satisfactory closure of the defect.

When part of the inguinal ligament has been lost in the failure of the original repair, reconstruction of this structure and closure of the large femoral canal with a suture or sutures obtained from the fascia lata will be required in order to obtain a satisfactory result.

The use of silk for sutures and ligatures in every case is a worth while addition to the technic, particularly since these wounds are prone to fill up with serum when catgut is used.

Patients in whom a recurrent femoral hernia has been repaired should be kept in bed longer than in the case of the primary repairs. The additional time is to be determined by the nature of the repair required in each individual case.

1213 Medical Arts Building.

PARENTERAL ADMINISTRATION OF A WATER-SOLUBLE COMPOUND WITH VITAMIN K ACTIVITY

4-AMINO-2-METHYL-1-NAPHTHOL HYDROCHLORIDE

EDWARD R. ANDERSON, M.D.

JOHN E. KARABIN, M.D.

HERBERT UDESKY, M.D.

AND

LINDON SEED, M.D.

CHICAGO

A water-soluble compound with vitamin K activity, 4-amino-2-methyl-1-naphthol hydrochloride, has been synthesized by Doisy and his associates.[1] The oil-soluble vitamin K, 2-methyl-1,4-naphthoquinone, has been found very active when administered orally,[2] but since it is relatively insoluble in water, it is inconvenient for parenteral use. Parenteral administration of vitamin K has several advantages over oral administration. By the oral route, there may be a lack of absorption due to intestinal obstruction, paralytic ileus or some other intestinal complication. By the parenteral route the vitamin may be given to patients who are unable to take it orally because of nausea or vomiting. In the treatment of hemorrhagic disease of the newborn, for which vitamin K is very effective, the parenteral method is especially indicated. When the substance is administered parenterally it is not necessary to give bile salts. With oral administration bile salts must be present in order to assure absorption of the vitamin.

From the Department of Surgery of the University of Illinois College of Medicine and the Cook County Hospital.

1. Doisy, E. A.; MacCorquodale, D. W.; Thayer, S. A.; Binkley, S. A., and McKee, R. W.: The Isolation, Constitution and Synthesis of Vitamin K, Science **90**:407, 1939. Thayer, S. A.; Binkley, S. A.; MacCorquodale, D. W.; Doisy, E. A.; Emmett, A. D.; Brown, R. A., and Bird, O. D.: Vitamin K Potencies of Synthetic Compounds, J. Am. Chem. Soc. **61**:2563, 1939.

2. (*a*) Anderson, E. R.; Karabin, J. E.; Udesky, H., and Seed, L.: The Oral Administration of Synthetic Vitamin K, to be published. (*b*) Rhoads, J. E., and Fliegelman, M. T.: The Use of 2-Methyl-1,4-Naphthoquinone (a Synthetic Vitamin K Substitute) in the Treatment of Prothrombin Deficiency, J. A. M. A. **114**:400 (Feb. 3) 1940. (*c*) Macfie, J. M.; Bacharach, A. L., and Chance, M. R. A.: Vitamin K Activity of 2-Methyl-1,4-Naphthoquinone and Its Clinical Use in Obstructive Jaundice, Brit. M. J. **2**:1220, 1939.

Before the synthesis of the water-soluble compound, phthiocol had been found to contain vitamin K activity (Almquist and Klose [3]) and had been used intravenously. Phthiocol was isolated by Anderson and Newman [4] from the pigment of the tubercle bacillus and was found to be 3-hydroxy-1,4-naphthoquinone. Smith [5] and also Butt, Snell and Osterberg [6] reported good results after using phthiocol intravenously. Ansbacher and Fernholz [7] found the potency of this compound to be about one hundred times less than that of vitamin K concentrates. However, the oil-soluble 2-methyl-1,4-naphthoquinone was almost as active as the vitamin K concentrates in pure form. Thus, phthiocol was not a satisfactory parenteral substitute for vitamin K. Rhoads and Fliegelman [2b] used 2-methyl-1,4-naphthoquinone, 1 mg. in 10 cc. of physiologic solution of sodium chloride, intravenously in 1 case, with a good response, but its relative insolubility made it inconvenient for general use. Sharp and his associates [8] found that 4-amino-2-methyl-1-naphthol hydrochloride contains vitamin K activity. Infants and patients with obstructive jaundice,[9] as well as experimental animals, responded satisfactorily within twelve hours.[10]

The soluble synthetic compound used in this investigation was 4-amino-2-methyl-1-naphthol hydrochloride;[11] for convenience in this report it will be designated as vitamin K_s. The prothrombin determinations were done by the Smith method. The value for plasma prothrombin is reported as the per cent of prothrombin clotting activity and represents the per cent of normal.

---

3. Almquist, H. J., and Klose, H. H.: The Antihemorrhagic Activity of Certain Naphthoquinones, J. Am. Chem. Soc. **61**:1923, 1939.

4. Anderson, R. J., and Newman, M. S.: The Chemistry of the Lipids of Tubercle Bacilli, J. Biol. Chem. **101**:773, 1933.

5. Smith, H. P.; Ziffren, S. E.; Owen, C. A., and Hoffman, G. R.: Clinical and Experimental Studies on Vitamin K, J. A. M. A. **113**:380 (July 29) 1939.

6. Butt, H. R.; Snell, A. M., and Osterberg, A. E.: Phthiocol: Its Therapeutic Effect in the Treatment of Hypoprothrombinemia Associated with Jaundice; a Preliminary Report, Proc. Staff Meet., Mayo Clin. **14**:497, 1939.

7. Ansbacher, S., and Fernholz, E.: Simple Compounds with Vitamin K Activity, J. Am. Chem. Soc. **61**:1924, 1939.

8. Sharp, E. A.; Emmett, A. D., and Kamm, O.: The Vitamin K Activity of 4-Amino-2-Methyl-1-Naphthol and 4-Amino-3-Methyl-1-Naphthol, J. Biol. Chem. **133**:285, 1940.

9. Sharp, E. A.; Vonder Heide, E. C., and Good, W. H.: Vitamin K Activity of 2-Methyl-1.4-Naphthoquinone and 4-Amino-2-Methyl-1-Naphthol in Hypoprothrombinemia, to be published.

10. The work was done in the research laboratory of Parke, Davis & Company.

11. The material used in the clinical experiments herein reported was furnished by Parke, Davis & Company, who have called it vitamin K_s; each ampule contains 1 mg. of the substance dissolved in 1.4 cc. of water.

Eighteen patients were given this compound parenterally to study its therapeutic effect; it was hoped thereby to compare its efficiency with that of the insoluble vitamin K previously used. Seventeen patients responded favorably, while 1 did not. The patient last mentioned had fatal acute yellow atrophy of the liver due to antisyphilitic therapy. Apparently the entire liver had been destroyed, this destruction producing a condition comparable to that observed after complete hepatectomy. Since the liver is necessary for the manufacture of pro-

TABLE 1.—*Jaundiced Patients Who Received Vitamin K₅*

Patient	Diagnosis	Jaundice		Prothrombin Activity	After Vitamin K
		Duration	Degree		
A. N.	Acute yellow atrophy of liver	2 weeks	4	37%	37%
A. R.	Metastatic carcinoma	6 weeks	4	50%	100%
C. K.	Carcinoma of ampulla of Vater	2 months	4	67%	100%
S. V.	Carcinoma of pancreas	2 months	4	79%	100%
C. P.	Carcinoma of pancreas	1 month	3	61%	90%
M. C.	Carcinoma of pancreas	2 months	3	64%	94%
S. D.	Carcinoma of pancreas	3 weeks	3	65%	100%
J. W.	Carcinoma of pancreas	3 weeks	2	65%	100%
A. W.	Acute cholecystitis	2 weeks	2	75%	100%
O. M.	Cholelithiasis	2 weeks	1	62%	100%
W. C.	Catarrhal jaundice	2 days	1	55%	98%
J. L.	Toxic hepatitis	12 days	1	50%	95%
H. M.	Choledocholithiasis	10 days	1	64%	94%
A. M.	Cholelithiasis	1 month	1	61%	100%

Grade: 1, slight; 2, moderate; 3, severe; 4, very severe.

TABLE 2.—*Nonjaundiced Patients Who Received Vitamin K₅*

Patient	Diagnosis	Prothrombin Activity	After Vitamin K
J. S.	Cholelithiasis	80%	100%
A. K.	Empyema of gallbladder	67%	100%
J. W.	Cirrhosis of liver	67%	100%
C. A.	Carcinoma of rectum	69%	85%

thrombin through vitamin K, this negative result was to have been expected. Of the 18 patients, 13 (table 1) had hypoprothombinemia due to jaundice, and 5 (table 2) had hypoprothombinemia due to other conditions. No patients had any signs of bleeding, for the hypoprothrombinemia was not allowed to reach dangerous levels.

The average initial dose was 2 to 3 mg. given intravenously and followed by 3 mg. daily as a maintenance dose. Some patients received 1 mg. daily and responded favorably, while others needed larger doses. The intramuscular dosage used was the same. However, this method of administration was not as satisfactory as the intravenous route. The case of O. M. (chart 1), a patient with cholelithiasis and jaundice due to secondary hepatitis, illustrates the intravenous therapy followed.

The level of plasma prothrombin before operation was 84 per cent. On the first day following cholecystectomy the level dropped to 62 per cent. Two milligrams of vitamin K_5 was given intravenously, and in one-half hour the level reached 86 per cent. In two hours it was 100 per cent. Twenty-four hours later it had dropped to 71 per cent. At this time 1 mg. was given intravenously three times a day. The prothrombin level returned to normal and remained there with this regimen. H. M. (chart 2), a patient with jaundice due to a common stone, illustrated the more gradual response by the intramuscular

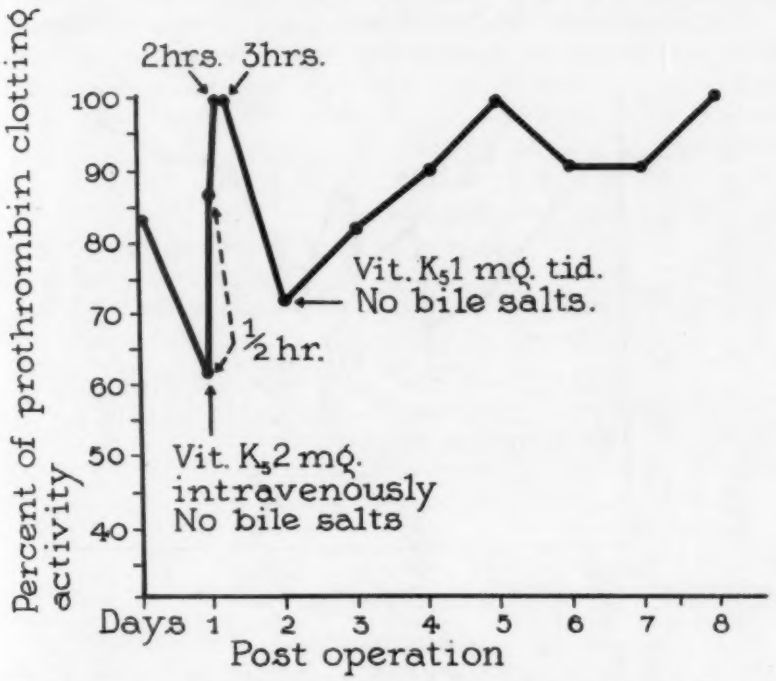


Chart 1.—Record in the case of O. M., a patient with cholelithiasis with jaundice. Note the sharp return of prothrombin clotting activity to normal within two hours after intravenous administration of 2 mg. of vitamin K_5 on the first postoperative day. A day later the prothrombin activity had dropped to 71 per cent.

A daily dose of 1 mg. three times per day brought the level back to normal, where it was maintained. The patient had jaundice—presumably secondary to hepatitis, since there was no stone in the common duct.

route. The prothrombin level on the first day following a choledochostomy was 83 per cent; on the second, 70 per cent, and on the third, 64 per cent. Three milligrams of vitamin K_5 was given intramuscularly. In one hour the level was 75 per cent; in two hours, 76 per cent, and in three hours, 80 per cent. The per cent then dropped to

74 in four hours. Three milligrams of vitamin K_3 was given intramuscularly with 1 Gm. of bile salts by mouth. In one hour the level increased to 78 per cent; in two hours, to 84 per cent, and in three hours, to 86 per cent. The level again dropped in twenty-four hours to 67 per cent. Vitamin K_2 (2-methyl-1,4-naphthoquinone), 2.4 mg. with 1 Gm. of bile salts, was given by mouth. In twenty-four hours the level was 88 per cent; in forty-eight hours, 90 per cent, and in seventy-two hours, 96 per cent. This level was maintained. However, the patient was improving generally, and the two factors were probably responsible for the continued oral improvement.

The response to the intravenously given vitamin K–active compound occurred within three-quarters of an hour to one and one-half

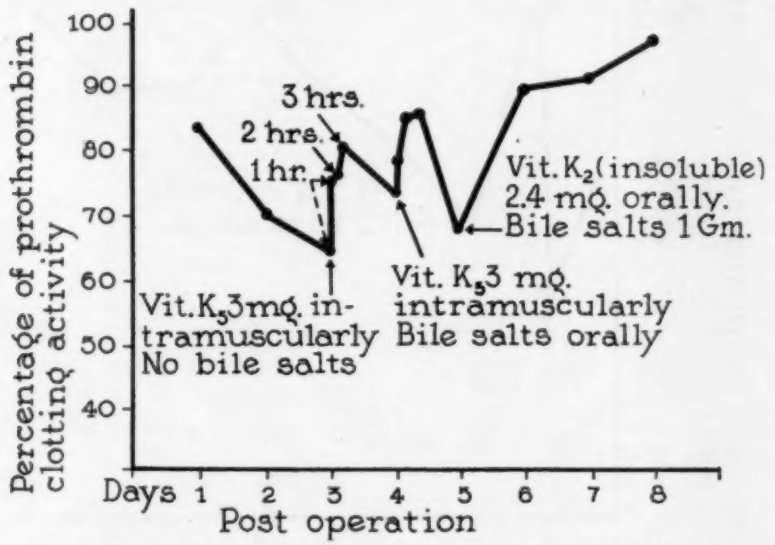


Chart 2.—Record in the case of H. M., a patient with choledocholithiasis and jaundice. Choledochostomy was performed. Note the drop in prothrombin clotting activity after operation, with a gradual response to intramuscular injection of 3 mg. of vitamin K_3. No difference in response to the vitamin K_3 is noted with the administration of bile salts orally.

Oral administration of vitamin K_2 maintained the level within normal limits.

hours. Patients with relatively good hepatic function showed normal levels of plasma prothrombin within two hours after the administration of the active compound. However, patients with damaged livers often did not reach a normal level of plasma prothrombin until twelve hours had elapsed; others never reached normal levels, in spite of the prolonged therapy. As has been stated, A. N., a patient with a severe degree of jaundice due to acute yellow atrophy of the liver, did not respond to 6 mg. of the active compound given intravenously. This

indicates the importance of the liver in the formation of the plasma prothrombin. Thus, clinically the patients with severe damage to the liver did not give as good a response to the compound or maintain the level of plasma prothrombin as well as those with relatively good hepatic function. In one-half hour, O. M. (chart 1) showed a rapid response to 2 mg. given intravenously. The plasma prothrombin activity rose 24 per cent in one-half hour and 38 per cent in two hours. Other patients did not respond so quickly. In the case of C. K. the value rose 20 per cent in three quarters of an hour; in the case of C. A., 16 per cent in one hour; in the case of M. C., 28 per cent in one hour, and in the case of A. W., 15 per cent in one and one-half hours.

The dose of the vitamin K–active compound needed to maintain the plasma prothrombin varied with the individual patient. Maintenance doses could be estimated only by repeated prothrombin determinations. One milligram three times a day or 3 mg. in one dose was a satisfactory amount for maintenance (case of O. M., chart 1). After an initial dose of 3 mg. the concentration of plasma prothrombin rose to normal, but it frequently dropped again on the following day. We thought that the effect of this water-soluble vitamin K compound was as good as that of the oil-soluble active compound. Broun [12] used an initial 5 mg. dose of the vitamin K_s in 1 instance and obtained a good response which extended over several days.

No bile salts were necessary for a response to vitamin K_s given parenterally. No toxic manifestations were observed. As much as 6 mg. at one dose was given intravenously without harm to the patient. Several patients were given 0.33 Gm. (a 5 grain capsule) of bile salts orally for each milligram of vitamin K_s given intravenously. No difference in the response or in the maintenance requirement was observed. Therefore, bile salts need not be given when parenteral vitamin K therapy is used. However, the value of bile salts for patients who are not secreting bile salts into the intestinal tract must be recognized. The deficiency of bile salts may produce adverse effects in the body economy not related to prothrombin.

Intravenous administration of the synthetic soluble active compound has an advantage over administration by the intramuscular route. The response is quicker, and the effect is greater. Chart 1 illustrates results of using the intravenous route. The response was quick, and a normal level of prothrombin was reached within two hours. Chart 2 illustrates a gradual and incomplete response to intramuscular therapy. From our observations the potency of 4-amino-2-methyl-1-naphthol hydrochloride, the water-soluble synthetic compound seems equal in effect

12. Broun, G. O., in discussion on Sharp, E. A.: Vitamin K Activity of 2-Methyl-1,4-Naphthoquinone, J. A. M. A. **114**:439 (Feb. 3) 1940.

to the oil-soluble synthetic compound. The advantage of vitamin K_s over the water-insoluble vitamin K compound therefore lies in the fact that it can be given at any time, even though the patient may be vomiting and unable to retain or absorb drugs given orally.

We were unable to obtain an appreciable elevation of the level of plasma prothrombin above normal with the parenteral active compound. In several instances a 5 per cent increase above the normal level of plasma prothrombin was observed.

SUMMARY

A water-soluble synthetic compound with vitamin K activity for parenteral use was effective in 17 of 18 patients with hypoprothrombinemia. The patient who failed to show a response had acute yellow atrophy of the liver.

Intravenous administration is superior to intramuscular. The ease of administration should determine whether the drug is to be given orally or intravenously.

The intravenous dose is approximately 2 to 3 mg. daily. The response occurs within three quarters of an hour to one and one-half hours after administration. Patients with relatively good hepatic function showed normal plasma prothrombin levels within two hours, while patients with damaged livers reached normal levels in twelve hours or did not reach normal levels in spite of prolonged vitamin K_s therapy.

Clinically, patients with severe damage of the liver did not give as good a response to the vitamin K–active compound or maintain the level of plasma prothrombin as well as patients with relatively good function of the liver.

The maintenance dose of the vitamin K-active compound varies with the individual patient and can be found only by repeated prothrombin determinations. No toxic effects were observed following doses up to 6 mg.

Bile salts are not necessary for the action of the drug when it is given either intravenously or intramuscularly.

The effect of vitamin K_s is no greater than that noted after the use of the water-insoluble vitamin K compound, but vitamin K_s has the distinct advantage that it can be given at any time in the preoperative or postoperative course, regardless of whether the patient is able to take and absorb drugs by mouth.

ROLE OF INTRALUMINAL OBSTRUCTION IN THE PATHOGENESIS OF ACUTE APPENDICITIS

HARRY KOSTER, M.D.
AND
ARTHUR SHAPIRO, M.D.
BROOKLYN

In the past few years several papers [1] on acute appendicitis have stressed the role of increased intraluminal pressure as a factor in the development of the lesion. Since the maximum intraluminal pressure can never exceed the pressure at which the appendix will empty into the cecum, studies were undertaken on the initial resistance to perfusion of unselected normal and diseased appendixes both in situ and after excision.

METHOD

Eighteen appendixes were studied in situ. After the appendix was delivered into the wound it was held by an Allis clamp applied to the mesentery as it joined the tip, and a transfusion cannula was passed into the lumen of the organ through the tip. The cannula was then connected to an easily controlled pressure system (water) with a glass tube side arm in which the fluid level could be observed. The pressure was slowly raised, and the height at which the level could no longer be sustained was considered the maximal initial perfusion resistance pressure. Appendectomy was then performed.

Twenty-five appendixes were studied after being removed without clamping or handling. The mesoappendix was picked up with an Allis clamp, and the appendix was delivered into the wound. The mesoappendix was tied and cut. Another ligature was tied around the cecum about 1 cm. from the base of the appendix. A purse-string suture was placed in the cecum. The appendix was excised with that portion of the cecum beyond the ligature, and the stump was inverted by the purse-string suture. The tip of the appendix was then cut off, and a cannula

From the Crown Heights Hospital.

1. (*a*) Wangensteen, O. H., and Dennis, C.: Ann. Surg. **110**:629, 1939. (*b*) Bowers, W. F.: Appendicitis, with Especial Reference to Pathogenesis, Bacteriology and Healing, Arch. Surg. **39**:362 (Sept.) 1939. (*c*) Dennis, C.; Buirge, R. E., and Wangensteen, O. H.: Surgery **7**:372, 1940. (*d*) Wangensteen, O. H.: Proc. Inst. Med. Chicago **12**:266, 1939. (*e*) Wangensteen, O. H.; Buirge, R. E.; Dennis C., and Ritchie, W. P.: Ann. Surg. **106**:910, 1937. (*f*) Wangensteen, O. H., and Bowers, W. F.: Significance of Obstructive Factor in Genesis of Acute Appendicitis: Experimental Study, Arch. Surg. **34**:496 (March) 1937.

was tied a few millimeters into the lumen. The initial resistance to perfusion was then determined in the same way as in the unexcised specimens. All specimens were then examined histologically.

TABLE 1.—*Initial Resistance to Perfusion of Unexcised Appendixes*

Case	Perfusion Resistance Pressure, Cm.	Fecalith	Condition of Organ	Operation	Comment
1	Over 150	+	Acute gangrenous appendicitis	Appendectomy	.....
2	Over 150	+	Acute suppurative appendicitis	Appendectomy	.....
3	Over 150	+	Acute suppurative appendicitis	Appendectomy	.....
4	134	+	Normal	Appendectomy	.....
5	106	—	Acute suppurative appendicitis	Appendectomy	.....
6	82	—	Normal	Suspension of uterus; appendectomy; salpingo-oophorectomy, right	.....
7	62	—	Normal	Herniotomy; appendectomy	.....
8	56	+	Acute gangrenous appendicitis	Appendectomy	.....
9	52	+	Acute suppurative appendicitis	Appendectomy	.....
10	45	—	Normal	.....	.....
11	43	—	Normal	Resection of broad ligament and veins of left foot; presacral nerve resection; appendectomy; partial resection of right ovary	.....
12	40	—	Acute suppurative appendicitis	Appendectomy	.....
13	38	—	Acute suppurative appendicitis	Appendectomy	.....
14	35	+	Normal	Sterilization; suspension of uterus; appendectomy; hemorrhoidectomy	Pressure sustained to 35 cm.; fecalith expelled; pressure dropped to 15 cm.
15	20	+	Normal	Appendectomy; partial resection of right ovary	.....
16	16	—	Acute gangrenous appendicitis	Appendectomy	.....
17	5	—	Acute suppurative appendicitis	Appendectomy	.....
18	4	—	Normal	Resection of terminal ileum, cecum, ascending colon and half of transverse colon	.....

Since the question under consideration is the role of increased intraluminal pressure as a factor in the pathogenesis of acute appendicitis, for the purpose of this discussion all appendixes which showed no evidence of acute inflammation are classified as normal.

RESULTS

In table 1, cases in which the appendix was perfused in situ are listed in the order of the initial resistance to perfusion. There were 3 appendixes which resisted perfusion despite a pressure above 150 cm. of water. All 3 showed appendicitis and contained fecaliths which were obstructing the lumen. At the other extreme, however, was a case (case 17) of acute suppurative appendicitis in which there was an initial perfusion resistance of only 5 cm. of water. Between these two extremes, cases of acute appendicitis were observed in which the resistance pressures were 106, 56, 52, 40, 38 and 16 cm. of water respectively.

On the other hand, the normal appendixes in this series were almost evenly interspersed between the inflamed ones, with pressures of 134, 82,

TABLE 2.—*Excised Appendixes*

Case	Pressure, Cm.	Cecum	Fecalith	Condition of Organ
19	150	Absent	+	Acute suppurative appendicitis
20	150	Present	+	Normal
21	150	Present	+	Acute suppurative appendicitis
22	150	Present	+	Normal
23	120	Present	+	Normal
24	150	Present	+	Normal
25	150	Present	+	Normal
26	120	Present	+	Normal
27	120	Present	+	Acute gangrenous appendicitis
28	110	Present	+	Normal
29	97	Present	—	Acute necrotizing appendicitis
30	95	Present	+	Normal
31	90	Present	+	Normal
32	90	Absent	—	Normal
33	76	Present	—	Normal
34	65	Absent	—	Acute gangrenous appendicitis
35	60	Present	—	Acute gangrenous appendicitis
36	57	Present	+	Acute gangrenous appendicitis
37	52	Present	—	Normal
38	44	Present	—	Normal
39	36	Absent	—	Acute suppurative appendicitis
40	36	Absent	—	Acute necrotizing appendicitis
41	35	Present	—	Acute gangrenous appendicitis
42	20	Absent	—	Acute gangrenous appendicitis
43	0	Present	—	Acute gangrenous perforative appendicitis

62, 45, 43, 35, 20 and 4 cm. of water. None of the normal appendixes sustained pressures as high as did those in cases 1, 2 and 3, in which acute appendicitis was present.

In table 2, cases in which the appendix was perfused after excision are listed in the order of the initial resistance to perfusion. In this group the absence of correlation between initial perfusion resistance pressure and acute inflammation is even more striking than with the appendixes perfused in situ. Of the 7 appendixes which resisted a pressure of over 150 cm. of water, 4 were normal and 3 were acutely inflamed. In the remainder of the table the normal and inflamed appendixes are fairly evenly dispersed, as they were in table 1.

In table 3 the cases in tables 1 and 2 are combined and arranged so that normal and acutely inflamed appendixes are separated and listed in the order of their initial resistance to perfusion. In all cases of

resistance to pressures of 110 cm. of water or more there were fecaliths obstructing the lumen. Of the 14 cases of this type, the appendixes in 7 were acutely inflamed, and those in the remaining 7 were not.

Of the appendixes in the remaining 29 cases, 7 had fecaliths. Of these, 4 showed acute appendicitis (57, 56, 52 and 20 cm. of water respectively), and 3 were normal (90, 35 and 20 cm. of water respectively). The remaining 12 were acutely inflamed, and 10 normal appendixes had no fecaliths.

In short, in the 43 appendixes in which the initial perfusion resistance pressure was measured there was no definite correlation between resis-

TABLE 3.—*Initial Resistance to Perfusion of Normal and Inflamed Appendixes*

Acute Appendicitis		Normal Appendixes	
Perfusion Resistance Pressure, Cm.	Fecalith	Perfusion Resistance Pressure, Cm.	Fecalith
150	+	150	+
150	+	150	+
150	+	150	+
150	+	150	+
150	+	134	+
150	+	120	+
120	+	110	+
106	−	95	−
97	−	90	+
65	−	90	−
60	−	82	−
57	+	76	−
56	+	62	−
52	+	52	−
40	−	45	−
38	−	44	−
36	−	43	−
36	−	35	+
35	−	20	+
20	+	4	−
16	−		
5	−		
0	−		

tance to perfusion and the presence of acute inflammation. There was, however, good correlation between resistance to perfusion and the presence of fecaliths.

COMMENT

Wangensteen and Dennis [1a] have presented data from which they have concluded: "It appears likely that the chief inciting agency in bringing about appendicitis in man is obstruction of an appendix in which the mucosa possesses the normal secretory capacity."

Since our data on the initial perfusion resistance pressure of the appendix as an index of intraluminal obstruction is not entirely consistent with their conclusions, it seems worth while to review their evidence in detail.

They studied 22 patients in whom the appendix was ligated at its base and exteriorized coincidentally with the performance of colostomy. In 5 of these there developed intraluminal pressures of 90 cm. of water

or more in a manometer connected to a cannula tied into the tip of the appendix. All of these appendixes had "good mucosa" in the control sections and gave microscopic evidence of "acute diffuse appendicitis" in sections taken after high intraluminal pressure had developed.

Of the remaining 17 cases, there were 4 in which no sections were obtained before obstruction and 14 in which no sections were obtained after obstruction.

There were 3 in which sections were obtained after obstruction. In case 9, in which there developed a pressure of 42 cm. of water, there were no control sections, and the condition was reported as "mild acute diffuse appendicitis with exudate in the lumen" after obstruction. In case 17, in which a maximum pressure of 8 cm. of water developed, there was no mucosa in the control section, but serositis and a few leukocytes in the muscle layer were observed after obstruction. Curiously enough, in case 20, in which there was no mucosa in the control section, there were mucus and leukocytes in the lumen, but no intraluminal pressure developed. No description is given of the muscularis or of the serosa to distinguish the condition in this case from "acute diffuse appendicitis."

This is the sum total of the evidence which Wangensteen and Dennis present as "experimental proof of the obstructive origin of appendicitis in man."

A critical analysis of these data suggests the following considerations:

1. Since in 14 cases there were no histologic studies of the appendix after obstruction of the lumen, no conclusions can be drawn as to whether the histologic picture of "acute diffuse appendicitis" was present in these (control?) cases.

2. Since the amount of appendix exteriorized was too short for biopsy in 64 per cent of all the cases studied, or 82 per cent of the "control" cases, it seems not unlikely that the specimens taken from those appendixes reported to have acute diffuse appendicitis were taken very close to the point where the cannula was tied in. If this is so, the inflammatory change observed may well have been due to local injury of tissues in the neighborhood of the cannula and the ligature.

It becomes apparent, then, that from the data of Wangensteen and Dennis [1a] no definite conclusions can be drawn regarding the causation of appendicitis in man. This is particularly unfortunate, because such conclusions might have established the relation of appendicitis in the human being to the various types of appendicitis studied in animals by these investigators.[1]

Returning to the data presented in table 3, it is clear that a definite correlation does exist between high initial resistance to perfusion and the presence of fecaliths. It is possible, then, that in the 7 cases of acute

appendicitis in which high initial resistance to perfusion was found in the presence of an obstructing fecalith, secretion against obstruction may have played a role in the pathogenesis of the disease. On the basis of the available evidence this can be neither affirmed nor denied. However, in 13 cases in our series of 23 (table 3) in which acute appendicitis developed in appendixes with initial perfusion resistance pressures of 60 cm. of water or less, fecaliths were present in only 4. This suggests strongly that secretion against obstruction is not the only mechanism for the development of appendicitis.

On the other hand, the finding of 7 normal appendixes which had initial perfusion resistance pressures of 110 cm. of water or more and contained fecaliths is evidence that obstruction of the lumen does not necessarily cause appendicitis.

SUMMARY AND CONCLUSIONS

The "initial perfusion resistance pressure" was studied in 43 appendixes removed at operation and was correlated with the histologic observations.

Eighteen studies were done in situ and 25 immediately after appendectomy without clamping or handling of the organ.

The results were similar in the two groups and may be summarized together.

1. There is a definite correlation between high initial perfusion resistance pressure and the presence of fecaliths.

2. High initial perfusion resistance pressure due to obstruction by fecaliths may occur in the normal appendix.

3. While secretion against obstruction may be a factor in the pathogenesis of acute appendicitis, it is certainly not the only mechanism.

4. There was no definite correlation between resistance to perfusion and the presence of acute inflammation.

FIBROSARCOMA OF SOFT TISSUE PRODUCING REGIONAL CONCENTRIC BONE ABSORPTION

ORMAND C. JULIAN, M.D.
CHICAGO

The relation between the usual type of soft tissue sarcoma invading bone and the resulting bony change is well recognized. Such a lesion is commonly a fibrosarcoma of the fascial plane which grows with moderate rapidity and produces ragged absorption of adjacent bone.

The case recorded here is that of a very slowly growing fibrosarcoma of the soft parts of the forearm which first affected the bones seven years after onset of the tumor. The morphologic structure of the tumor was such that its malignant character was not suspected from examination of tissue removed at the first operation and was never definite on the basis of histologic observations alone. The unusual nature of the bone lesion resulting from the sarcoma, the great length of the history of the tumor, the difficulty encountered in making the pathologic diagnosis and the results of the conservative therapy used owing to this difficulty make this case worthy of an individual report.

REPORT OF A CASE

Mrs. E. B., a woman 43 years of age, was first seen at the Billings Memorial Hospital in January 1934. In 1922 she had fallen downstairs, hurting the left arm but sustaining no severe injury. One year later, in 1923, a small tumor was noted beneath the skin on the dorsal surface of the left forearm, about 3 inches (7.6 cm.) above the wrist. The tumor grew slowly during the following years until 1930, when the central portion became indented. In July 1930 a roentgenogram of the forearm, taken because of this tumor, showed, as compared with the same region of the other arm, a sloping reduction in diameter of 3 cm. of the shaft of the radius under the region of the tumor. No treatment was instituted, and very slow growth of the tumor continued, with only occasional slight discomfort in the region. A second roentgenogram, taken in June 1933, showed a more advanced diminution in circumference of the radius 2 to 3 inches (5 to 7.6 cm.) above the wrist (fig. 1).

On November 16 the patient sustained a fracture of both bones of the forearm at the level of the tumefaction. The arm was immobilized in a cast for eight weeks, but at the time of examination at the Billings Hospital, Jan. 26, 1934, the fractures were ununited.

Coinciding with the fracture level there was seen an indurated, umbilicated "soft tissue tumor" of the skin and subcutaneous tissue of the dorsum of the

---

From the Department of Surgery of the University of Chicago.

Presented before the Chicago Pathological Society, March 11, 1939.

This work was aided by a grant from the Douglas Smith Foundation for Medical Research of the University of Chicago.

Figure 1

(See legend on opposite page)

Figure 2

(See legend on opposite page)

forearm (fig. 2 *A*). In a roentgenogram (fig. 2 *B*) the radial fragments showed a 2 to 3 cm. segment of moderate concentric absorption of the shaft, centering on the fracture line. The ulnar fracture was at about the same level, but the fragments showed no absorptive change.

Open reduction with bone grafting was performed on January 26 by Dr. D. B. Phemister. With the tumor of the soft parts excluded from the field, the radius was exposed at the fracture level through an anterior incision. The shaft was found to be reduced nearly to one half of its normal diameter at the fracture site. There was no peripheral callus or evidence of tumor on the fragment ends. A small amount of callus between the bone fragments was curetted out, and a strip of periosteum 3 cm. long was cut from the upper radial fragment. Two full thickness bone strips from the right tibia were placed as onlay grafts, one anteriorly and one laterally. After closure of this wound a large biopsy specimen was taken of the depressed lesion of the skin and subcutaneous tissue. A cast was applied.

Pathologic Report.—The tissues were examined microscopically. The section of skin and subcutaneous tissue taken from the depressed lesion contained a layer of thin squamous epithelium with little cornification. The corium and the subcutaneous tissue were completely replaced by fibrous tissue. This fibrous tissue in different areas varied in maturity and in cellularity. In the more densely cellular regions (fig. 3) the cells were large and were spindle and oval shaped, with dark chromatin and distinct chromatin-perichromatin differentiation. No mitotic figures were present. The cells were arranged in streams and whorls. No palisading of the nuclei was evident. The callus from between the fragments was histologically mature collagenic connective tissue, with several spicules of living cancellous bone. The periosteal strip had a thin attached layer of cortical bone. Along the entire surface was an interrupted line of periosteal new bone and more superficially a layer of condensed collagen, thickest near the fracture, which represented the callus stimulated by the fracture. Dense, mature fibrous tissue (fig. 4) was seen in contact with the cortical bone at the end away from the fracture. This tissue had produced a broad, smooth indentation in the cortex. Examination of the cortical edge from this region toward the fracture disclosed, beneath the traumatically stimulated callus and new bone, numerous lacunar spaces resembling Howship's lacunae in form but containing no giant cells. The last-mentioned changes apparently represented the pathologic process which produced the concentric absorption noted in the roentgenograms taken before the fracture. No tissue resembling tumor was present.

The cast was removed on March 26, eight weeks after operation, and a roentgenogram showed bony union of the fracture. A mild degree of concentric absorption of the ulna was then noted at the healed fracture site. The "biopsy wound" of the depressed lesion had healed, but after a few months it broke down.

Fig. 1.—Roentgenogram showing reduction in the circumference of the radius (June 1933).

Fig. 2.—*A*, photograph of the arm two months after fracture with resulting nonunion, showing the tumor of the forearm and its depressed center. *B*, fractures of both bones, sustained in November 1933. The radial fracture is through the narrowed region shown in figure 1.

On November 7 a deep ulcer was present in the indurated zone on the dorsum of the forearm. The hard edge of the ulcer was narrow and the base crusted. Biopsy material from the base and edge was taken on this date.

A section taken from this biopsy tissue contained a segment of skin which was normal to the abrupt, unelevated edge of a shallow ulcer. The floor was

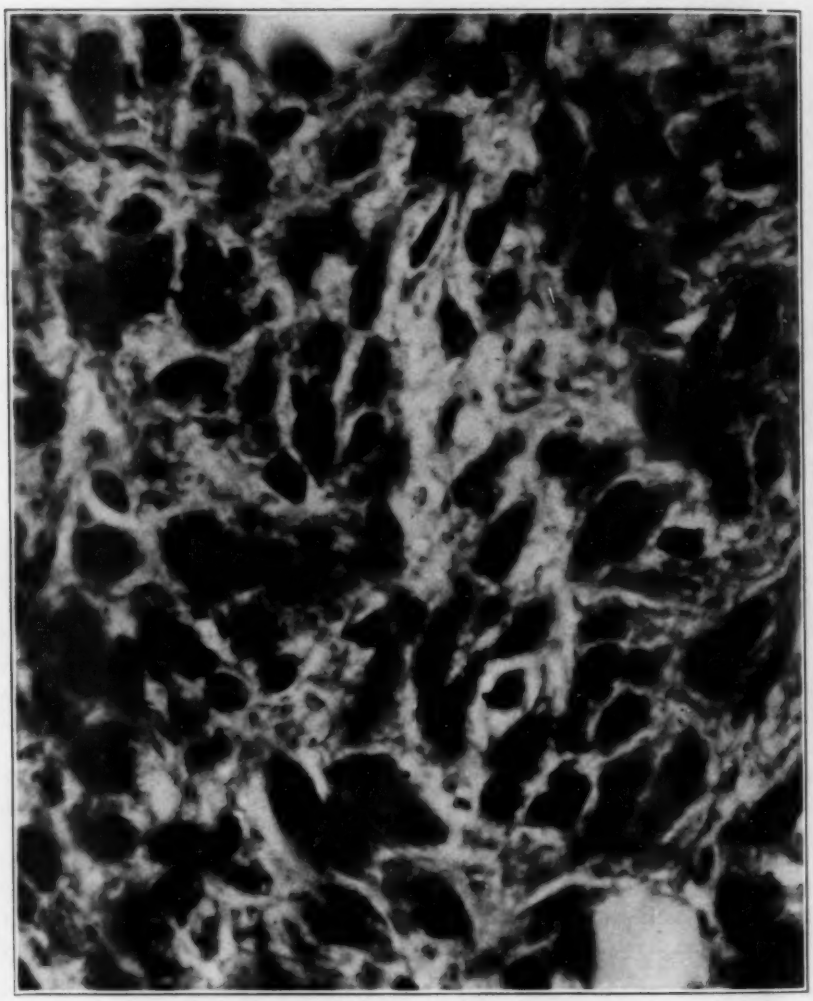


Fig. 3.—Photomicrograph of a less mature area in the scarred subcutaneous tissue. The tissue was taken at the time of the first bone grafting.

covered by a superficial thin layer of necrotic tissue, beneath which the subcutaneous tissue was replaced by very immature connective tissue. The cells of the tissue were round and spindle shaped and formed invading whorls and strands

occupying the immediately subcutaneous tissue and extending in some areas into the subcutaneous fat. One definite mitotic figure was seen (fig. 5).

Because the results of biopsy suggested malignancy, excision of the entire field of the ulcer and the indurated areas was done on December 3. A full thickness pedicle graft was applied to the area after excision of the lesion. At operation

Fig. 4.—Photomicrograph of the proximal end of a piece of periosteum, showing dense, nontumorous fibrous tissue encroaching on the cortex of the radius.

grayish, nodular tissue was found invading the subcutaneous tissue. It extended down to the ulna and was dissected off the tendons and ulna, some tissue being left behind.

Pathologic Report.—Gross Observations: The specimen consisted of a piece of skin and subcutaneous tissue 5 cm. square, in the center of which was a granulating ulcer 1.5 cm. in diameter, having narrow, hard walls. The underlying subcutaneous tissue was indurated. There were several fragments of hard, nodular

Fig. 5.—Photomicrograph of an area in the subcutaneous tissue, showing a single mitotic figure (from the biopsy specimen of skin taken on Nov. 7, 1934).

trabeculated tissue which had been dissected from the subcutaneous and intertendinous regions. Two nodules measured 2 by 1.5 by 1.5 cm. and 3 by 1 by 0.5 cm. respectively. On cross section they presented a smooth, pale, densely fibrous surface.

Microscopic Observations: Sections were taken of the skin, the edge of the ulcer and various regions in the nodular tissue beneath. The epidermis was of

normal thickness in the region examined. There was a mild inflammatory infiltration in the narrow necrotic border of the ulcer. The superficial layers of the dermis showed densely collagenous scar tissue with sparse normal spindle cell nuclei. However, below the dermis a different type of connective tissue was seen. Here the cells, which formed nodular whorls and streams, varied tremendously in size, density of chromatin and shape. These cells in the subcutaneous tissue infiltrated from below the more superficial "normal" scar tissue in several places. Only three or four mitotic figures were seen in several sections of this invading type of tissue. However, the pleomorphism, immaturity and hyperchromatism suggested a slowly growing sarcoma. Sections of the deeper nodules contained the same pleomorphic tissue.

The wound healed by the end of January 1935, and no sign of recurrence was observed grossly. However, in March several scattered nodules suggesting sarcoma appeared in the skin and subcutaneous tissues over the side and front of the radius. No axillary metastases could be felt.

Fig. 6.—Roentgenogram taken in October 1936, showing the ulnar fracture in a region of marked concentric absorption. The radial graft is united. The soft tissue shadow shows the position of the full thickness skin graft.

Roentgen therapy was given from March to July, 1935. A single dorsal portal on the forearm received 5,038 r. By June the subcutaneous nodules had disappeared owing to the radiation therapy.

The patient remained free of symptoms and signs of recurrence until October 1936, when she began to have a recurrent painless clicking sensation in the ulnar side of the forearm at the site of the old fracture. A roentgenogram showed considerable sclerosis of the reconstructed radius. The radius was intact. The ulna was fractured in the center of a region of extensive concentric absorption (fig. 6). At this time several cutaneous nodules had recurred. Biopsy specimens

were taken and showed tissue of the same morphologic character as that seen in the edge of the ulcer already described.

Roentgen therapy (735 r) was given again at the end of October 1936. A roentgenogram taken in March 1937 showed further absorption of the ulnar fragments leaving a gap between the ends. The patient returned on May 24, complaining of pain in the forearm and loss of function of the wrist, the radius having been refractured through the grafted region. The patient was readmitted to the hospital for operation.

Operation was done on May 26. No callus was seen in the fracture region. The tissues about the bone were scarred, but there was no gross evidence of tumor. A 4 inch (10 cm.) tibial bone graft and several cancellous bone chips were placed at the fracture level. After closure of this wound a second incision was made over the back of the ulna. A fracture of the ulna with spindle-shaped absorption of the ends of the fragments was found. The periosteum, which was thickened over the lower fragment, was reflected from both fragments. Beneath the periosteum of the lower fragment there was grayish tissue which looked like a thin layer of sarcoma. This was excised. No tumor tissue was found in the corresponding region of the upper fragment, but a strip of periosteum from here and several pieces of callus-like tissue from the fracture line were taken for examination. A whole thickness tibial graft and cancellous bone chips were applied at the fracture level.

Microscopic examination was made of all tissue removed. The bone chips and callus from the radius showed no tumor. The section of periosteum from the ulna showed various changes compatible with a weak attempt at healing of the fracture. The callus which was seen in the section showed only slight beginning calcification and no ossification. There was no definite evidence of sarcoma tissue in the region, which represented a segment of periosteum of the upper fragment 1½ inches (3.7 cm.) long. The section made of the gray tissue found on the distal ulnar fragment contained tissue similar to the infiltrating tissue seen in the biopsy specimen previously studied. The cells showed enlargement and variation in size and definite hyperchromatic staining of the nuclei. No mitotic figures were seen in the sections studied.

When the cast was bivalved, on July 26, an ulcer of the skin 5 cm. in diameter was found to have developed medial to the pedicle skin graft, exposing the deep fascia. Clinically, fibrous union of the fracture and grafts had occurred. A roentgenogram showed the grafts and fragments in good position, but there was no definite bony union. The ulcer remained unhealed despite removal of the pressure caused by the cast, and dead portions of the tibial grafts of the radius and ulna were sequestrated during the following months. Two small nodules recurred on the back of the hand in March 1938; biopsy specimens were taken.

Pathologic Report.—Gross Observations: The specimen consisted of a spindle-shaped piece of skin and attached subcutaneous tissue. Nothing abnormal could be seen on the surface of the skin, but on palpation one could feel a nodule in the subcutaneous tissue which seemed to be less than 0.5 cm. in diameter.

Microscopic Observations: The section was through skin and subcutaneous tissue. In the depth of the subcutaneous tissue there was a small, roughly round lesion. This lesion was composed of large, somewhat atypical cells having round and oval nuclei. The nuclear pattern was distinct in most of the cells. There were no mitoses. The central portion of the lesion had been replaced by a pink-staining hyaline tissue which continued peripherally for a short distance into the cellular portion of the nodule as pink-staining fibers. There were no giant cells

in the lesion. The periphery was surrounded by a collar of normal lymphocytes. There was no capsule to suggest that the tissue was an invaded lymph node. There was no invasion of the surrounding tissue. The general appearance of this lesion was that of a chronic granuloma. The central portion, however, on close exami-

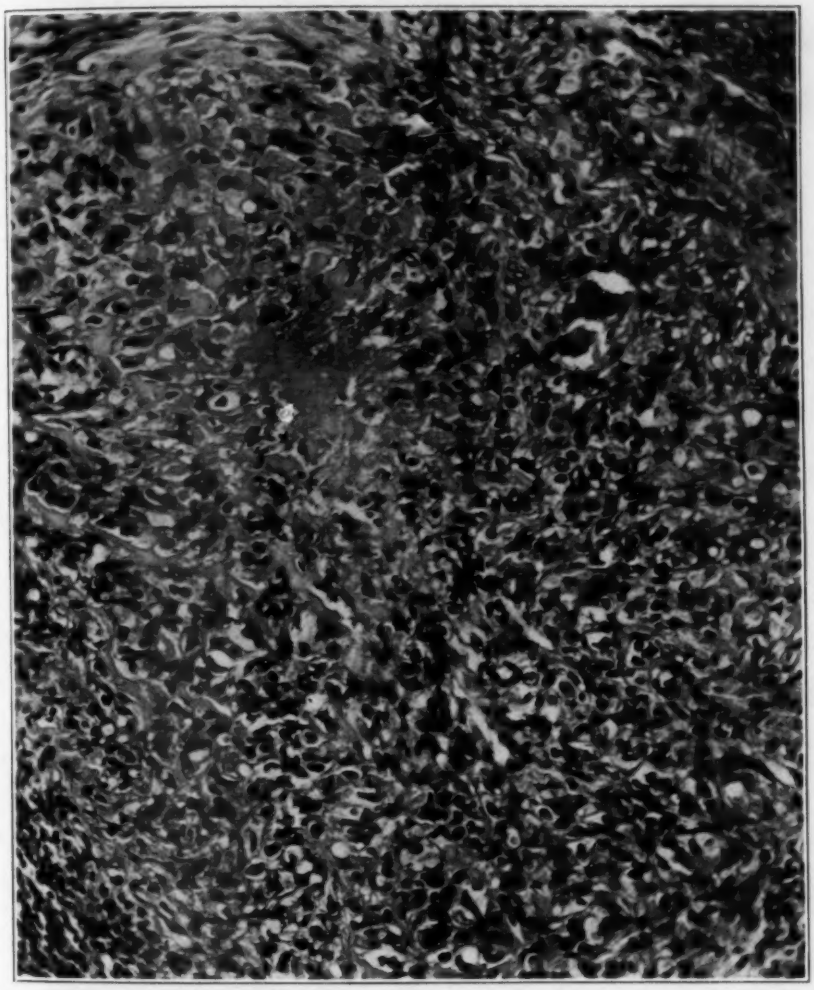


Fig. 7.—Nodule in the subcutaneous tissue of a nodule from which a biopsy specimen was taken in March 1938. Note the central hyalinized portion and the continuation of the hyaline fibers out into the peripheral portion of the lesion.

nation was seen to be composed of hyaline fibers (fig. 7) and not of necrotic material. Acid-fast stain of this section showed no organisms.

The hand was now almost completely functionless, and a roentgenogram taken in June 1938 revealed a complete fracture of the radius at the same level as the previous fractures. The radial graft distal to the fracture line was absent, having

been sequestrated. The graft proximal to the fracture was firmly united to the shaft.

On July 5 the arm was amputated 1½ inches (3.7 cm.) above the condyles of the humerus.

Pathologic Report.—Gross Observations: The specimen consisted of the left forearm, the elbow and the distal 3 inches (7.6 cm.) of the humerus. Four centimeters above the radiocarpal joint there was an ulcer measuring 4 by 2.5 cm. over the dorsal radial surface of the forearm. Its edges were shiny, white, scarred and firmly attached to the underlying bone. The floor of the ulcer was a surface of bone. This bone appeared to be dead. Over the interosseous area, forming the ulnar border of this large crater, there was a full thickness graft which was firmly healed in place except where it bordered on the ulcer already described and in two small areas on the lateral side, where breaking down had occurred. The subcutaneous tissue about the ulcer and graft was markedly fibrous and was frozen to the underlying bone. There was a linear scar at the level of the condyle of the radius in which there was a hard shotty nodule measuring about 0.75 cm. Three smaller nodules were palpable in the skin of the dorsum of the hand. These were small and superficial, projecting above the skin. The forearm was narrowed at the level of the ulcer.

The bones of the forearm, when stripped of all soft tissue, presented very irregular surfaces. The radius was fractured directly beneath the soft tissue ulcer. This fracture showed no attempt at healing. Extending from this fracture proximally for a distance of 5 cm. there was a solidly united portion of bone graft. The distal one fourth of this graft appeared white and dead, a sharp transverse groove being present at the junction between the living and the dead bone. The distal fragment of the radius was narrowed as it approached the fracture line; the fragment appeared to be alive.

The ulna was markedly thinned for a distance of about 4 cm., centering on the point opposite the radial fracture. In this narrowed region the medullary cavity of the bone was completely obliterated by dense bone. The remaining grafted bone had remained united, and its boundaries were indistinguishable on the uneven surface of the ulna.

Microscopic Observations: The sections included the various nodules on the back of the hand, the nodule found in the cutaneous scar at the elbow, portions of the scar tissue from the region of the fractures and sections of the bone constituting the distal 18 cm. of the radius and the distal 9 cm. of the ulna.

The radial segment was sectioned in two parts. They showed the articular end, a long portion of the shaft with fracture and a totally fused portion of a bone graft on the proximal fragment. This graft represented the proximal segment of the first graft applied. Its distal end formed the proximal part of the bony floor of the ulcer crater seen in the gross specimen. Microscopically, the articular cartilage of the distal radial articulation was replaced completely by fibrocartilage. The articular cortex of the bone was thin and in some places incomplete. The atrophic cancellous bone of the distal end of the shaft was composed of thin, widely spaced trabeculae. The marrow here was fatty. The cortex at the distal end was thin but rapidly became thicker proximally, where it assumed a grossly abnormal architecture. The bone retained its lamellar pattern, but the trabeculae were thick and branching. Some of these were composed of acellular bone. The fatty marrow near the distal end of the radius was invaded by irregular masses of anaplastic round and oval cells arranged in whorls and streams (fig. 8). These

cells had laid down a small amount of collagen and had stimulated a fibrous tissue reaction about them of normal adult fibroblasts. One or two mitotic figures were seen in the sections examined. This same tissue was present in one large area on the surface of the radius over the region of invaded medullary canal.

Fig. 8.—Photomicrograph of a section of the radius in the amputated specimen. The medullary cavity is invaded by tumor tissue. Several spicules of dead cancellous bone are in the field.

The graft was seen to be firmly fused with the shaft of the proximal fragment of the radius. The graft itself was composed of cortical bone, some of which was acellular. The medullary spaces of the graft were filled predominantly by fibrous tissue, but a few areas showed invasion by tumor. The distal end of

the graft, which was exposed in the soft tissue ulcer, was entirely dead bone. It showed the changes of acute osteomyelitis.

The ulnar segment resembled the radius in atrophy and regions of replacement of fatty marrow by tumor tissues. A portion of the bone in the region of

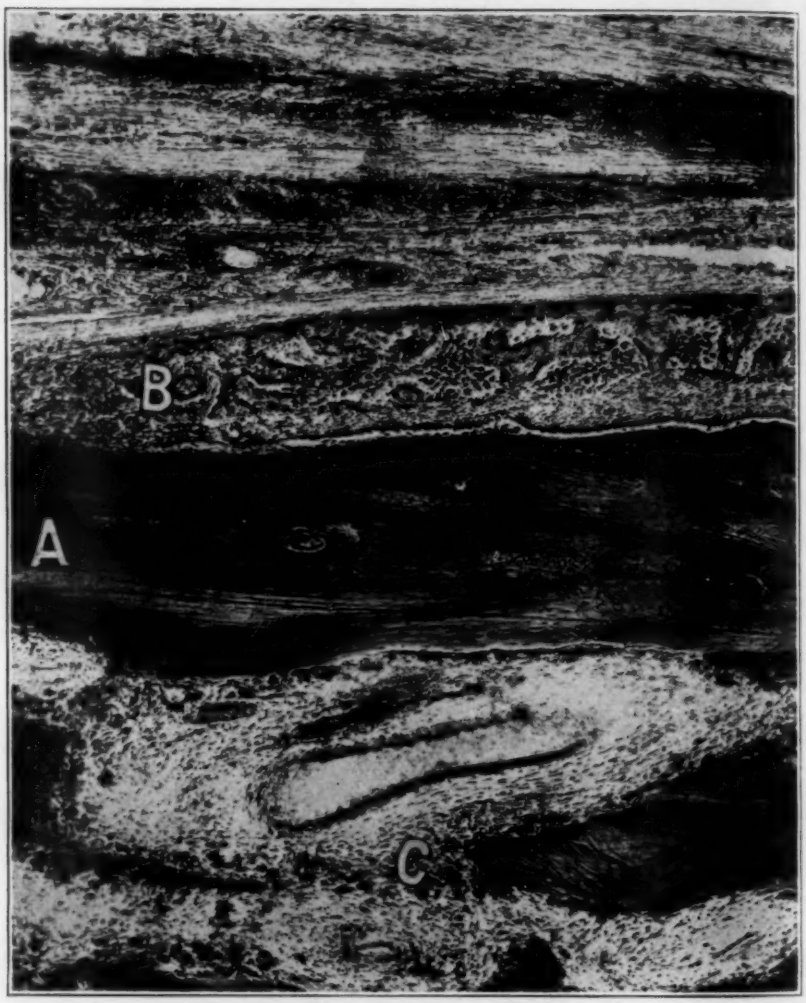


Fig. 9.—Photomicrograph of the cortex of the ulna and scar tissue on its surface taken from the amputated specimen. *A*, cortex of the shaft; *B*, periosteal new bone with tumor tissue in the intertrabecular spaces; *C*, fibrovascular tissue in the medullary spaces, showing marked giant cell absorption of bone.

the fractures and bone grafting contained no medullary cavity and appeared to represent a completely assimilated bone graft.

A short segment of periosteal new bone was present on one surface of the ulna (fig. 9). The tissue in the spaces between the trabeculae of this new bone was indistinguishable from the invading tumor tissue seen within the bone.

Sections of the cutaneous nodules showed small collections of sarcoma tissue in the superficial portion of the subcutaneous tissue. The tissues taken from the firm, scarred region about the radius and ulna contained very dense collagenous connective tissue with frequent infiltrates of tumor cells.

After operation, the humeral stump healed without event.

In January 1939, a small, nontender, firm nodule was noticed in the upper third of the scar in the right leg (fig. 10). This nodule was only 0.5 cm. in diameter, and because it was situated exactly in the incision it was considered to be scar tissue. However, its center became depressed, forming an umbilicated

Fig. 10.—Depressed surface of the tumor nodule which developed in the scar of the right leg.

though not ulcerated lesion. It was excised in March and revealed immature fibrous tissue similar to that seen in various other nodules and tissues already examined. This wound healed rapidly and at the time of this report shows no evidence of recurrence.

The tendency of this tumor to attack the skin was again observed in November, when a tiny nodule was found in the scalp. On excision the same low grade sarcoma tissue was seen. Several more nodules then appeared in the scalp and were treated in February 1940 by application of radium.

At the time of the present report the patient is free of local recurrence but is receiving roentgen therapy to the scalp to supplement the radium therapy.

COMMENT

The diagnosis in this case is slowly growing fibrosarcoma of the soft parts of the arm with an unusual type of concentric absorption of the bones. At first operation there was no gross or microscopic evidence of tumor at the fracture site or on the surface of the fragments.

The late appearance of a sarcomatous lesion in the wound in the leg is of great interest when one considers the sequence of surgical procedures of the operation during which the graft was taken from this region. The tumor of the soft parts had been excluded from the incision in the arm, and the only tissue handled which gave possibility of transplant was that about the radial fracture. This tissue showed neither gross nor microscopic evidence of malignancy. However, the fact that sarcoma tissue was almost certainly transplanted from the fracture site throws considerable doubt on this evidence.

The histologic structure of the cutaneous nodules, of the various tissues removed at operation and of the tissue invading the bones found after amputation was not in itself diagnostic of a malignant neoplasm but rather suggested an immature fibroma.

The possibility of a leprous infection was suggested by the roentgen appearance of the forearm, and one of the cutaneous lesions strongly suggested a chronic granuloma (fig. 8). Acid-fast stains of several of the tissues gave negative results.

The cause of the primary osteolytic process in the radius is obscure, though malignant disease was suggested by the transplant already described. It might be explained on a basis of vascular changes associated with the growth of a tumor in the immediate vicinity.

Osteolytic changes following trauma, with or without fracture, are well recognized. However, the so-called post-traumatic painful osteoporosis (Sudeck's atrophy) is most frequently seen in the hands and feet and takes the form of generalized moth-eaten osteoporosis of a region and not that of concentric absorption of a short segment of bone.

Recently, several cases reported by French authors[1] have been grouped as examples of essential osteolysis. In these cases there was very slowly progressive, usually concentric, absorption of a segment of the skeleton. In all except 1 the lesion was preceded by a history of trauma. In those in which the patients were operated on, no definite tumor was found. Bone grafting, done in 2 of these cases, gave results comparable to those obtained in my case. The lesion extended to the

1. Radulesco, A. D.: Un cas curieux d'ostéolyse compléte de l'extrémité inférieure, J. de radiol. et d'electrol. **21**:304, 1937. Mouchet, A., and Rouvillois, H.: Ostéolyse du bassiu d'origine indeterminé, ibid. **21**:263, 1937. Dupas, J.; Badelon, P., and Daydé, G.: Aspects radiologiques d'une ostéolyse essentielle progressive de la maine gauche, ibid. **20**:383, 1936.

graft in each case and produced concentric absorption. No vascular, endocrine, metabolic, hereditary or inflammatory factor was found in these cases, but a neoplastic process could not in any case be ruled out.

SUMMARY

A case covering a seventeen year history of slow concentric absorption of both bones of the forearm is presented. The diagnosis was made of soft tissue fibrosarcoma of very low grade malignancy. This was supported by the finding of cutaneous metastases. Other possible diagnoses—granuloma, notably leprosy, Sudeck's atrophy and so-called essential osteolysis—are discussed.

REVIEW OF UROLOGIC SURGERY

ALBERT J. SCHOLL, M.D.
LOS ANGELES
FRANK HINMAN, M.D.
SAN FRANCISCO
ALEXANDER VON LICHTENBERG, M.D.
BUDAPEST, HUNGARY
ALEXANDER B. HEPLER, M.D.
SEATTLE
ROBERT GUTIERREZ, M.D.
NEW YORK
GERSHOM J. THOMPSON, M.D.
AND
JAMES T. PRIESTLEY, M.D.
ROCHESTER, MINN.
EGON WILDBOLZ, M.D.
BERNE, SWITZERLAND
AND
VINCENT J. O'CONOR, M.D.
CHICAGO

KIDNEY

Anomaly.—MacKenzie [1] reported a case in which two separate clinical entities occurred in the same organ; namely, cystic inflammation of the pelvis and ureter and embryonic defect or lack of development that had occurred during intrauterine life.

Cystic disease of the urinary tract is primarily a disease of old age, occurring in persons of either sex from whom a history of disturbance or definite inflammation of the urinary tract can be elicited. The condition is reported to have occurred in several cases of double ureter, and, as in MacKenzie's case, it may occur in conjunction with congenital renal hypoplasia.

MacKenzie admitted that proliferative renal fibrosis and chronic pyelonephritis with interstitial and glandular degeneration as encountered in his case may be termed "renal atrophy." However, his assumption was that these changes represent secondary atrophy superimposed on hypoplasia, the latter being purely congenital in origin.

1. MacKenzie, D. W.: Renal Hypoplasia with Pyelitis Cystica and Ureteritis Cystica: Report of a Case and Review of the Literature, Tr. Am. A. Genito-Urin. Surgeons **32**:321-335, 1939.

Smith [2] reported 8 cases of horseshoe kidney. Two of the patients were not operated on; 2 underwent left nephrectomy for hydronephrosis; 3 underwent pyelolithotomy and 4 underwent division of the isthmus. Symptoms of pressure by the isthmus on the great vessels were exhibited by 2 patients. The need for investigation of the blood supply before division of the isthmus is done was emphasized. Smith did not advise division of the isthmus in patients who show no signs or symptoms of pressure on the great vessels but stated that such patients should have the benefit of repeated excretory urograms for a period of years to insure against the development of renal damage. If such patients are found to have stones or hydronephrosis, the isthmus should be divided when the operation for stone or hydronephrosis is done.

Tuberculosis.—Randall [3] reported a case of bilateral renal tuberculosis of thirty-seven years' standing and of known and proved existence from the time the patient was 18 years old to the time of the report, when the patient was 55. At the time of writing the patient weighed 190 pounds (86 Kg.) and had every appearance of enjoying perfect physical health. Actually no vesical symptoms were observed, but at intervals of two to three months the passage of renal calculi caused fever, pain and occasionally hematuria.

In explaining the unusual latency and delay of progress of the disease, Randall stated that the responsible organism may be of low virulence or the resistance of the host extremely high. But he expressed the belief that there is a basically distinct difference in renal balance when the two organs, each of normal development, are simultaneously affected to an equal degree by disease or obstruction. While he was studying by means of urograms the silent unilateral renal death which occurs when malignant tumor of the bladder gradually occludes one ureteral orifice, Randall observed cases in which the renal response was different when both ureters were equally obstructed from the response noted when only one ureteral orifice was occluded. When one ureteral orifice was occluded, its kidney quietly ceased to function, and the opposite kidney carried on bodily functional needs without symptomatic complaint or physical change. But in cases in which the two ureters were about equally involved, pictures of extreme response to necessary bodily demands were observed, and bilateral hydroureters and hydronephroses of increasing degrees pictured nature's efforts and the renal response to fight to the last ditch, so to speak, and to the ultimate point of renal counterbalance before allowing these vital organs to fail.

---

2. Smith, G. G.: Surgery in the Horseshoe Kidney, Tr. Am. A. Genito-Urin. Surgeons **32**:73-83, 1939.

3. Randall, A.: Bilateral Renal Tuberculosis: A Unique Case, J. Urol. **43**: 35-38 (Jan.) 1940.

Randall expressed the belief that in the case of bilateral renal tuberculosis which he reported there was evidence of nature's compensatory efforts to carry on vital function when the two organs are equally involved, for it was impossible for either organ to shift its burden and functional responsibility to an uninvolved mate.

De Langre [4] reported a case in which renal tuberculosis was associated with cysts of the kidney in a girl 16 years of age. The patient had never had any renal or vesical symptoms until pyuria had made its appearance, one and a half years prior to her examination by De Langre, and it had been discovered in the course of a general examination for a state of fatigue. Mycobacterium tuberculosis subsequently was found in the patient's urine, and nephrectomy was performed for a tuberculous right kidney. Examination of the specimen revealed the presence also of a number of small cystic cavities and tuberculous granulations.

On the basis of what Roubier, Cibert and Barral wrote in 1935, only 6 cases of this kind appear to have been found in the literature. The case discussed here differed slightly from the others, in all of which there were true polycystic kidneys. The kidney in the present instance contained numerous cysts but was not much increased in size, and the cysts were small, being properly termed microcysts of the kidney rather than the type commonly observed in polycystic kidneys. The pathogenesis of such cysts, however, is not clearly understood. The designation "cysts of nephritis" is well suited to those found in the kidneys of elderly persons or in kidneys affected by chronic nephritis, but the patient in De Langre's case was very young and had never shown any signs of having had nephritis earlier. Nor does it appear that tuberculosis was the cause of the cysts in De Langre's patient. All kinds of transitional stages could be observed, in fact, from cysts with smooth walls, invested with endothelium, to cysts with granular seeds on the wall and cysts transformed into tuberculous caverns. The tuberculosis in this case was clearly secondary, grafted on the cysts. The hypothesis of renal malformation, comparable to that of polycystic kidney, is much more acceptable as an explanation of the mode of formation of such cysts than is tuberculosis.

The other kidney of De Langre's patient appeared to be perfectly sound; it could not be palpated, and the pyelogram of the pelvis and calices showed them to be normal. The case confirms the conclusion reached by Roubier, Cibert and Barral that tuberculous lesions may become grafted on a preexisting polycystic kidney.

Keyes [5] discussed closure of the wound in the loin without drainage after nephrectomy for tuberculosis. Of the patients from whom he

4. De Langre: Tuberculose et kystes du rein, J. d'urol. **47**:138-140 (Feb.) 1939.

5. Keyes, E. L.: The Closure of the Loin Wound Without Drainage After Nephrectomy for Tuberculosis, Tr. Am. A. Genito-Urin. Surgeons **32**:33-37, 1939.

removed tuberculous kidneys and who survived and were followed for more than a year after operation, clamps had been left on the pedicle in 13. Four of these died of tuberculosis or renal insufficiency. Two of them were alive at the time of his report but had tuberculosis three and five years, respectively, after operation. One was active and well twenty-four years after operation. Seven were well when they were last heard from.

Of the 9 patients who underwent drainage without clamps and who were followed, 3 were dead of tuberculosis six, six and thirteen years, respectively, after operation. One had died of embolus. Two patients were alive and had tuberculosis five and twelve years respectively after operation, and 3 were well respectively eight, eleven and twenty years after operation.

Of the 23 patients treated by suture without drainage and followed, 10 were dead of tuberculosis, and 13 (more than half) were alive and well, at the time of the report.

Keyes stated that as a result of this experience he was impressed with the theory that after nephrectomy for tuberculous kidney the drain in the loin is a site of mixed infection and sinus formation in the loin. If the sinus opens after complete closure, this fact need not imply a tempestuous or a prolonged convalescence. Drained tuberculous wounds today do well; undrained wounds do better. Keyes expressed the belief that the results of modern atraumatic surgical technic will be better than his were since the last war; namely, 20 of 23 wounds in the loin closed without any drain whatever, and two sinuses formed, only one of which was a grave one. He stated that since surgeons are no longer timid about tying the renal artery, it is not necessary to be timid about closing the tuberculous loin.

Rathbun [6] stated that in all cases in which definite dense, hard strictures at the lower end of the ureter are present in the widely dilated ureter, complete ureterectomy should be done in addition to nephrectomy. There are some cases in which, for one reason or another, ureterectomy is rather a formidable procedure.

Rathbun expressed the belief that, no matter how careful aseptic technic may be, a certain number of bacteria are injected into every wound that is made. He wrote that nature takes care of such bacteria pretty well if allowed a reasonable chance to do so, but any incision in the loin is a highly traumatizing operation, with much bruising of the muscles and the like. Much serous exudate forms, and an outlet should be provided for such exudate; otherwise pressure necrosis may occur,

6. Rathbun, N. P., in discussion on papers of Randall, Howard and Keyes, Tr. Am. A. Genito-Urin. Surgeons **32**:42, 1939.

which sets the stage for infection. A simple cigaret drain inserted at the dependent angle of the incision and removed in forty-eight hours certainly should not do any harm, if one proviso is carefully observed: The dressing should be done with the same painstaking, aseptic technic that characterized the operation.

Deming [7] stated that there are three principles concerning incisions made in operations for tuberculous kidneys which should be kept in mind by urologic surgeons. The first is that all tuberculous tissue should be removed. Deming advocated removing the whole ureter with the kidney. The second is that protection of the incision should be afforded as far as injury to the walls is concerned. The third is a surgical principle which some surgeons forget—complete drainage of the incision. Not all incisions are dry. In cases in which the incisions are wet, a drain left in position for forty-eight or seventy-two hours relieves the wetness of the incision. General surgical experience tells the urologic surgeon that a dry incision heals far better than does a wet incision. Deming had not had any incision break down during the nine years prior to his discussion. The fact that resident surgeons in the service were able to obtain equally good results shows, Deming stated, that the three principles which he mentioned have some merit.

Colby [8] stated that for persons in whom the lower end of the ureter is strictured or in whom the ureter is much dilated he removes the ureter completely. In most of his cases, however, the ureter is simply treated by means of phenol and ligation.

None of the patients is operated on until he has been in the sanatorium for from two to six months. The low incidence of complete breakdown of incisions is in contrast to the incidence of such breakdowns among persons who have had their kidneys removed without the benefit of previous sanatorium treatment. Possibly, the same thing may be true of similar incisions in patients operated on for diseased ureter; in other words, the patient's resistance to this disease is brought to a considerably higher level by means of sanatorium treatment. Patients placed in a sanatorium previous to operation certainly do better after operation. In only 1 instance in Colby's experience in the last six or seven years was it thought necessary to remove a ureter which had been left in place after nephrectomy. This ureter was removed because the patient had persistent pain in that side, and about 6 inches (15 cm.) of atrophied ureter was removed, in which there was no microscopic evidence of tuberculosis.

7. Deming, C. L., in discussion on papers of Randall, Howard and Keyes, Tr. Am. A. Genito-Urin. Surgeons **32**:44, 1939.

8. Colby, F. H., in discussion on papers of Randall, Howard and Keyes, Tr. Am. A. Genito-Urin. Surgeons **32**:43-44, 1939.

Tumor.—Wharton [9] stated that the diagnosis of early renal tumor must occasionally be based on the general clinical picture or on suggestive evidence, in spite of the absence of any single infallible sign.

When there is presumptive evidence of renal tumor, the proper treatment is exploration.

Clinical data must be accurate and interpreted with judgment, in order that mistakes may be avoided. It is, however, a more serious error to postpone an operation until the situation is clear than it would be to perform an exploratory operation and find no hypernephroma. The decision requires the exercise of fine judgment, and the surgeon who saves lives occasionally will explore and find no tumor. Generally, however, some pathologic condition is present which will explain the bleeding; such bleeding rarely is idiopathic.

It is usually impossible to distinguish a benign cyst from a hypernephroma, and it is usually unsafe to presume that such a distinction can be made.

Preoperative irradiation also may confuse the picture in a questionable case and should be reserved for proved tumors.

The lumbar route is preferable to the transperitoneal route for surgical exploration of a kidney. The transperitoneal operation is indicated in cases in which the presence of a tumor has been established and in which the situation lends itself to this procedure.

Barney [10] reported a case in which extrarenal hypernephroma afflicted a white man 70 years of age. General examination, including the making of pyelograms, showed that a huge, partially calcified tumor was occupying the lower pole of the right kidney. At operation it was found that the tumor extended up under the costal margin. The tumor was somewhat necrotic and was markedly adherent to the lower pole, from which, however, it could be separated. The kidney was not removed. The diagnosis was hypernephroma. The patient died four years after the operation, at which time necropsy disclosed no evidence of metastatic disease.

Cahill [11] elaborated on two points in Barney's paper concerning extrarenal "hypernephromas." First, the term "hypernephroma" has been applied to tumors of a definite gross and microscopic appearance. These tumors definitely and frequently occur in the kidney, and they have been reported as a rare occurrence in the adrenal body and in

9. Wharton, L. R.: Hypernephromas That Are Too Early to Diagnose, J. Urol. **42**:713-719 (Nov.) 1939.

10. Barney, J. D.: Extrarenal Hypernephroma, Tr. Am. A. Genito-Urin. Surgeons **32**:47-56, 1939.

11. Cahill, G. F., in discussion on papers of Barney, Kretschmer and Smith, Tr. Am. A. Genito-Urin. Surgeons **32**:85-87, 1939.

adrenal rests. Adrenal rests are found in the retroperitoneal tissues anywhere from the adrenal region to the testes or the ovary.

In a careful review of roentgenograms of all the renal tumors which had been seen in the Squier Clinic, it was found that calcification shadows occurred in the tumor in 22 per cent of cases. The first published discussion of renal calcification was a paper on renal tumors written by Braasch. At that time, based on results obtained at the Mayo Clinic, Braasch thought that calcification permitted a rather favorable prognosis. However, Cahill stated that a review of reported cases led to the opposite conclusions. A study of large numbers of tumors characterized by calcification will show more conclusively whether calcification of the tumor has any special effect on the prognosis. Calcification apparently is secondary to necrosis or hemorrhage. Cahill also has found that calcification is a valuable sign in distinguishing solitary cysts from round tumors, depending on the appearance of the calcified deposits.

Nowlin [12] reported 2 cases in which 2 brothers died of malignant tumor of the kidney. One brother was 67 years old and had undergone left nephrectomy, with recovery. Death occurred two months later from pulmonary metastasis. The histologic picture was typical of hypernephroma. In the case of the other brother, 62 years old, the diagnosis was made by cystoscopy and urograms. He refused all treatment and died shortly afterward from pulmonary metastasis.

Calculi.—Gutierrez [13] drew attention to the vital problems presented by bilateral nephrolithiasis, which in the past was frequently regarded as definitely inoperable but for which the indications for operation today are known to be wider and the prognosis more favorable than ever before. Nevertheless, determination of the indications for surgical intervention and the operative management is still a difficult and serious task, requiring experience and the knowledge gathered from observation of many individual patients. There is no single rule; each patient must be treated according to the particular conditions present.

The frequency of bilateral nephrolithiasis is greater than has been supposed; it has been variously estimated to constitute from 6 to 20 per cent of all instances of renal stone. The mortality rate is more than double that accompanying the unilateral condition, the destructive pathologic process having always the tendency to destroy the entire renal parenchyma, with consequent complete loss of function. In addition, the maintenance of latent infection ultimately leads to anuria and terminal uremia.

---

12. Nowlin, P.: Familial Tendency to Renal Carcinoma, J. Urol. **43**:654-655 (May) 1940.

13. Gutierrez, R.: Surgical Treatment of Bilateral Nephrolithiasis, Urol. & Cutan. Rev. **43**:642-652 (Oct.) 1939.

Early diagnosis is of great importance, but unfortunately the silent course of the disease in many cases causes the patients to come late for surgical relief, and the fact that the well advanced lesion is bilateral may make a fatal outcome inevitable. Nevertheless, in every case in which nephrolithiasis is of moderate extent, the condition should be considered potentially operable. Cases in which no operation is permissible are those in which renal function is inadequate or in which the urogram reveals the presence of too little cortical parenchyma for restoration of function. Even in such cases, however, exception must be made of certain cases of calculous anuria and far advanced pyonephrotic kidney, since emergency nephrostomy performed for drainage may succeed in prolonging somewhat the life of the patient.

Among the most important types of bilateral nephrolithiasis are those in which: (1) a stone lies in the pelvis of each kidney; (2) both stones are of the staghorn type; (3) there is a stone in each kidney and one in one ureter; (4) the stone is in an anomalous kidney, perhaps a double kidney, on one side and in a normal kidney on the other; (5) the nephrolithiasis is in polycystic kidneys, and (6) the stone is in a horseshoe kidney. In addition to those, many other types exist, each of which calls for experienced judgment in the choice of an operation.

Diagnosis is made by roentgenograms, pyelograms and cystoscopic examination. Functional tests are essential, as are also an estimation of the amount of urea in the blood and the making of cultures. A roentgenogram which depicts the kidney as normal does not exclude the presence of certain types of stones, which may be revealed only by a filling defect in the pyelogram.

Gutierrez reported 6 of his own cases to illustrate the different problems that may be encountered in the surgical management of the condition under discussion. These bring out the importance of proper preliminary preparation and adequate postoperative treatment of the patient to attain permanent cure and to prevent recurrence. Two of the cases show the clinical confusion which exists in diagnosis, especially when secondary anemia is the conspicuous symptom, with the stone running a silent course. Three cases (reported through the courtesy of colleagues) illustrate bilateral coraliform stones afflicting patients who came too late, because of too advanced disease and poor general condition, for operation. These 3 cases serve to emphasize the need of recognition of such conditions before they become hopeless.

The surgical risk and operative mortality rate depend on the judgment of the surgeon as to the advantageous time to operate, a judgment which should be based on renal function, the urea content of the blood and the general condition of the patient. The right choice of a procedure, the rapidity with which it is carried out, the type of anesthesia employed and the preoperative and postoperative care all will play a part in the outcome.

The fundamental principle in these cases is to operate whenever it is possible, first on the better kidney, with a view to having this organ in good condition in the event that it is found advisable or practicable to perform nephrectomy on the other kidney. For the same reason it is always best to operate first on the kidney in which the stone is free in the pelvis and is smaller and hence more easily removed by simple pelviolithotomy or nephrolithotomy. The power of a kidney to regain its function is so remarkable that the principle of conservation is essential in treatment of surgical conditions arising in this system. The duty of the surgeon to save a kidney whenever possible cannot be too strongly stressed.

When a kidney contains multiple calculi, has fairly good function and is not pyonephrotic, it is important, after it has been surgically exposed, to examine it fluoroscopically and to make roentgenograms to rule out definitely the possibility that stones or fragments may have been left in any of the calices or in the renal parenchyma, since these "forgotten fragments" not only actually jeopardize the condition of the kidney but stimulate recurrence and infection, which may result in the formation of a subsequent renolumbar fistula and in the persistence of renoureterovesical infection.

When renal calculi are larger than the normal caliber of the ureter and cannot pass spontaneously or by means of cystoscopic and ureteral manipulations, the most common operations required are pelviolithotomy, nephrolithotomy, pelvionephrolithotomy, nephrostomy, calicystectomy, heminephrectomy, nephrectomy and ureteronephrectomy, the choice depending on the type of lesion and the condition of the individual patient.

For obtaining curative results, the maintenance of lumbar drainage after any type of conservative operation on an infected lithiasic kidney by means of a supplementary nephrostomy tube is of the greatest practical importance. Furthermore, mere surgical removal of the stones from the kidneys by no means constitutes adequate treatment for calculi, since in the modern concept of the relief and cure of these conditions postoperative care for the purpose of securing drainage, relieving infection and preventing recurrence is one of the most important requisites.

Oppenheimer [14] recommended operative roentgen control (roentgen study of the surgically exposed kidney) as a valuable aid in the operative treatment of renal calculi. In his series, in 29 (34 per cent) of 85 examinations performed under operative roentgen control, stones or fragments of stones which could not be palpated or found without the roentgenogram were located by means of it and were removed.

14. Oppenheimer, G. D.: Evaluation of Roentgenography of Surgically Exposed Kidney in Treatment of Renal Calculi, J. Urol. **43**:253-264 (Feb.) 1940.

Kearns [15] stated that in many instances the recurrence of stones is preventable if proper study is applied to causative factors. Application of the definite advances achieved in chemotherapeutic measures designed to combat infection alone will result in a diminution in the incidence of reformation of stone.

A fundamental requisite to proper preventive treatment lies in the administration of vitamin A, augmented by all the other vitamins for their synergistic action. Partial deficiency of vitamin A is a real possibility as a factor in the development of recurrent stones. Some persons have capricious appetities, and others have an aversion to certain essential foods. Still others, because of their economic status, are deprived of these elements; in some, faulty assimilation results in varying degrees of deficiency.

During the thirteen years prior to the time of his report, Kearns recommended vitamin A in the diet for the prevention of recurrence of urinary calculosis. During this period, among all the calculous patients observed by him, it was possible to follow a group of 164 who adhered to their dietary instructions. This group included all patients who either passed stones spontaneously or underwent surgical or cystoscopic removal of stone. It included also patients with and without infection. There were only 8 instances of recurrence, or 4.8 per cent. A large number included those who were uninstructed, who failed to follow treatment or who were untraced. An intake of water of 60 fluidounces (about 1,800 cc.) daily is advisable to promote mechanical flushing and to insure ample water for solubility of urinary solids.

Trauma.—Domrich [16] reviewed cases reported in the literature of blunt traumatism of the kidney. He reported 55 cases and came to the following conclusions:

The only indications for primary nephrectomy are severe hemorrhage and extensive destruction of the kidney. In a number of cases in which conservative treatment is carried out surgical intervention will be necessary later. Bleeding is the most serious complication and infection the second most severe. Half of Domrich's patients whose kidneys did not heal had infection of the urinary tract later. Hydronephrosis, ureteral stricture and renal stones are the most common late complications of trauma. The stones apparently form around a nucleus of a blood clot. Nephrectomy is the most common operation. Perinephritic abscess which develops after hemorrhage may be drained and the kidneys left intact. Patients who have undergone nephrectomy usually obtain good results and are capable of doing their usual work. Indus-

15. Kearns, W. M.: The Prevention of Recurrence of Urinary Stone, J. Urol. **43**:598-610 (April) 1940.

16. Domrich, H.: Die stumpfen Nierenverletzungen und ihre Folgezustände, Ztschr. f. Urol. **33**:337-381, 435-465 and 521-541, 1939.

trial accident commissions and insurance companies should take this lack of long-continued symptoms into consideration in the adjustment of these cases.

Denervation.—Wildbolz [17] reviewed the literature on denervation of the kidney and discussed the results of his own experiments on dogs and also his clinical experience. He found that all the nerves to the renal parenchyma enter at the renal hilus. A denervated kidney eliminates a greater number of solid substances than does a healthy kidney. Clinically, the best method for denervation of the kidney is mechanical destruction of the nerves situated along the renal hilus. Wildbolz and Schneider showed that this can be done by animal experimentation. They found also that the flow of blood through the denervated kidney is 60 to 100 per cent higher than the flow of blood through a normal kidney. The effects of denervation last about six months.

Wildbolz also discussed the clinical results obtained in 61 cases. Denervation is useful only in those cases in which the underlying pathologic process can be cleared up during the six months in which the effect of denervation persists. Results are most satisfactory in cases of acute nephritis, which endangers the life of the patient by anuria or which may develop into chronic nephritis. Results are also good with hematurias of unknown origin. If, after surgical exploration, conditions are found in the kidney other than those expected, denervation should not be done, since it might possibly cause harm. This operation is definitely not a panacea for diseases of the kidney, but in its proper place it is a very useful procedure.

Aneurysm of the Renal Arteries.—McClelland [18] discussed aneurysm of the renal artery, of which trauma is the most common cause. A history of a previous injury or of contusion of the kidney usually is obtained, and later the signs and symptoms of the aneurysm appear. Arterial disease itself, causing degenerative changes, such as those associated with arteriosclerosis, periarteritis nodosa and syphilis, favors the formation of aneurysm.

Hematuria, pain, swelling and pulsation are the usual symptoms. There may be a slowly growing swelling in the flank. The growth is progressive and is accompanied with a dull throb over the mass. In a few instances a systolic bruit occurs over the mass and is diagnostic. Hematuria may appear weeks or months after the original injury.

Mathé stated that in only 7 of 56 reported cases was the condition diagnosed or suspected before death of the patient or operation. The roentgenogram is of great value. A ring shadow denser at its periphery

17. Wildbolz, E.: Ueber die Enervation der Niere, Schweiz. med. Wchnschr. **69**:825-829 (Sept. 16) 1939.

18. McClelland, J. C.: Aneurysm of the Renal Artery, Tr. Am. A. Genito-Urin. Surgeons **32**:169-175, 1939.

and situated at the hilus of the kidney is diagnostic. A pyelogram will show this shadow to be entirely separate from the pelvis or the major calices. It may be situated directly between the two major calices.

Thirty-five of 56 untreated patients died. Seventeen of 21 patients operated on were saved from certain death by removal of the kidney. Death occurs as a result of exsanguination and is usually caused by hemorrhage into the peritoneal cavity, around the kidney or into the urinary tract. Most of the patients die within a year from the onset of the swelling and pain.

Operation should be performed as soon as the aneurysm is suspected. The true aneurysm is treated by nephrectomy through an incision in the flank.

Infection.—Nesbit and Dick [19] stated that acute staphylococcic infections of the kidneys are relatively common. They are hematogenous in origin, the lesion being cortical and showing a tendency to heal promptly and completely.

Costovertebral pain, tenderness and fever are constant. The urine rarely contains pus, but the stained sediment reveals cocci. Secondary bacillary infection of the urine frequently occurs. The disease tends to run a stormy, although self-limiting, course, ending in complete recovery.

Surgical complications occur in approximately 10 per cent of cases, and the appearance is heralded by an increase in symptoms and signs. These complications, perinephric abscess and carbuncle of the kidney, are readily diagnosed at onset and should be treated by immediate drainage. Pulmonary complications of perinephric suppuration occur in 16.5 per cent of cases and often cause delay in diagnosis and treatment of the underlying pathologic lesion. Such complications regress under adequate treatment of the underlying subdiaphragmatic disease.

Abscess.—Jeck [20] reported 2 cases of large solitary abscess of the kidney. In only a few of these cases was the condition recognized preoperatively.

One of Jeck's patients was a man in whose case a tentative diagnosis of perinephric abscess was made. No free pus was found around the kidney. A fluctuating region was detected on the posterior curvature of the kidney and was opened. Two fluidounces (59 cc.) of heavy pus was evacuated.

In the other case the patient, a woman 27 years old, had marked tenderness in the left costovertebral angle. She had a definite sensation of fulness in the left loin. The kidney was exposed through an oblique

19. Nesbit, R. N., and Dick, V. S.: Acute Staphylococcal Infections of the Kidney, J. Urol. **43**:623-636 (May) 1940.

20. Jeck, H. S.: Large Solitary Abscess of the Kidney, J. Urol. **43**:28-34 (Jan.) 1940.

incision in the loin. The perinephric fat was very pale. The kidney itself also was very pale and appeared to be somewhat enlarged and rather firm. In the upper half of the kidney, on the posterior surface, well up under the ribs, was felt a fluctuating region about the size of a half-dollar, and while an attempt was being made to separate the upper pole, a finger was accidentally passed into this region, which immediately released a large quantity of yellowish, fairly liquid pus. The cavity from which the pus came was then explored digitally and apparently did not communicate at all with the renal pelvis. As far as could be estimated, the cavity had contained 3 to 4 fluidounces (88 to 118 cc.) of pus, and its walls were smooth.

A history of boils, carbuncles, paronychia, acute osteomyelitis, infected teeth, infected tonsils or other, similar foci of infection is of the utmost importance in establishing a diagnosis when other signs point to the kidney. With all such infections chills are usually found to be followed by the spiking type of increased temperature, pain in the loin of the affected side, more or less tenderness about the infected kidney, a moderately high leukocyte count, a low erythrocyte count with a low hemoglobin percentage and, finally, a rather marked loss in weight.

The treatment of large solitary abscess of the kidney is incision and drainage. On exposure of the kidney when abscess is suspected, if the abscess is not at once apparent, decapsulation of the kidneys should be performed, for, as Beer has pointed out, a solitary abscess or multiple abscesses may not be discovered until the true capsule of the kidney is removed.

Perirenal Abscess.—Hamer [21] stated that perinephritic abscess of extrarenal origin in most cases is the result of blood-borne infection to the renal cortex followed by cortical abscess with extension to the perirenal tissues. The source of the infection is some remote focus, such as a carbuncle, a boil, an infected wound, or an infection of the respiratory tract. Calculous disease and tuberculosis are the most common causes of perinephritic abscess of renal origin.

The infecting organism is usually one of the pyogenic coccus group.

The chief symptoms are pain and tumor in the loin, with costovertebral tenderness, fever of the septic type and severe leukocytosis.

Diagnosis in the early stages is most difficult. Urologic study is of value in cases of primary renal disease, but in most cases of the metastatic condition positive urologic evidence is lacking.

Roentgenograms may show a clouded renal region, obscuration of the border of the psoas muscle and scoliosis.

---

21. Hamer, H. G.: Perirenal Abscess, Surg., Gynec. & Obst. **70**:595-597 (Feb. 15) 1940.

The value of thorough drainage is emphasized. With the metastatic type of abscess simple drainage is usually followed by prompt recovery. For the type of abscess which is secondary to existing renal disease nephrotomy or nephrectomy may be required. Nephrectomy usually should be postponed until drainage has relieved the patient of his sepsis.

Nephrostomy.—Moore [22] presented a method providing for immediate nephrostomy with the continued use of the same tube as an internal support of the ureter but without the undesirable feature of prolonged external nephrostomy drainage. This method should encourage prolonged splinting of the ureter after any type of operation on the ureteropelvic junction, since the tube can be retained without disturbance of the patient in any way. This procedure was used for 5 patients operated on for hydronephrosis. Moore also described a tube designed to eliminate the inadequacies of the ordinary rubber tube.

Nephrotomy.—Higgins and Glazier [23] discussed the relative merits of the scalpel and the high frequency current as employed in nephrotomy. In their experimental work they studied histologically the relative amount of primary hemorrhage, the intrarenal extravasation of blood along the lines of incision, the infarction of renal tissue and the relative rate of healing.

They concluded that less primary hemorrhage occurs when extensive nephrotomy is performed with the cutting current than when a scalpel is used. Extravasation of blood into the renal tissue adjacent to the line of incision is greater when the scalpel is used. There is less infarction adjacent to the line of incision when the high frequency current is employed. Healing is more or less similar with the two methods.

Nephropexy.—Young [24] discussed his technic for nephropexy, in which he employs decapsulation and suture of rolled-up capsule to muscles of the back. His technic has a twofold purpose: (1) to decapsulate the posterior surface of the kidney, so that firm adhesions between the substance of the kidney and the muscles of the back may be secured, and (2) to utilize the rolled-up edges of the capsule on each side of the decapsulated region as strong material around which sutures can be passed which hold the kidney against the muscles of the back. The capsule is divided along the midline of the posterior surface of the

22. Moore, J. G.: A New Procedure for the Correction of Ureteropelvic Junction Obstruction, Pennsylvania M. J. **43**:631-634 (Feb.) 1940.

23. Higgins, C. C., and Glazier, M.: Relative Merits of the Scalpel and High Frequency Current in Nephrotomy: Experimental Study, Tr. Am. A. Genito-Urin. Surgeons **32**:9-18, 1939.

24. Young, H. H.: Nephropexy: A Technique Employing Decapsulation and Suture of Rolled-Up Capsule to Muscles of Back, J. Urol. **43**:20-27 (Jan.) 1940.

kidney. The capsule is stripped off and rolled up, and the sutures are placed along the inner border and then along the outer border. Heavy chromic sutures are used, and the needle is then passed up through the psoas muscle about 2 cm. from the bodies of the vertebra.

Results of Nephrectomy.—Hanley [25] reviewed the postoperative results of nephrectomy. There were, altogether, 213 histories. Of the 182 patients traced, 100 actually were examined clinically, and their renal function was estimated.

The 213 patients whose histories were reviewed were placed in six groups, according to the original diagnosis: hydronephrosis; pyonephrosis; calculus; tuberculosis; neoplasms, and an unexpected diagnosis, chronic nephritis.

There were 40 cases in which nephrectomy had been done for simple hydronephrosis without any evidence of infection, the operations having been performed from two to six years prior to the time of Hanley's report. Twenty-nine patients were traced. The operative mortality rate for the group was 5 per cent; 2 deaths occurred within a fortnight of operation. The total mortality rate was 10 per cent, there being 2 additional deaths within two years of operation, both of the patients dying in uremic coma. No more deaths had occurred in this group during the four years prior to the time of writing.

There were 44 operations for pyonephrosis during the previous two to six years. Only 4 patients were untraced. The operative mortality rate was 20.2 per cent, 9 patients dying within a month of operation, 1 from pulmonary embolism. The total mortality rate for this group was 34 per cent; 6 additional deaths occurred during the following two years. One of these deaths followed nephrolithotomy two years after nephrectomy. No more deaths occurred in this group during the four years prior to the time of writing.

There were 49 cases in which nephrectomy for calculus was performed during the two to seven years prior to the time of the report. Forty of the patients were followed up. The operative mortality rate was 6.1 per cent, with 3 deaths. The total operative mortality rate was 16.3 per cent, with 5 additional deaths, all of which occurred within two years of nephrectomy except the death of 1 boy, who died five years after operation for a nonrenal condition.

Nephrectomy was done in 48 instances for supposedly unilateral tuberculosis of the kidney and was performed during the two to eight years prior to Hanley's report. Forty-two of the patients were followed up. The operative mortality rate was 4.1 per cent, with 2 deaths. The total mortality rate at the end of six years had increased to 22.9 per cent.

25. Hanley, H. G.: The Post-Operative Results of Nephrectomy, Brit. J. Surg. **27**:553-566 (Jan.) 1940.

Nephrectomy was performed in 27 instances for new neoplasms. The operative mortality rate was 22.2 per cent, 6 patients dying within a week of operation. The total mortality rate within the next two years increased to 63 per cent, 9 patients dying within thirteen months of the operation and another 2 within two years of operation.

There were 5 patients whose kidneys after removal showed evidence of chronic nephritis, with no histologic evidence of any other pathologic process. The operative mortality rate was 20 per cent, with 1 death occurring eight days after operation. The total mortality rate was 100 per cent, all the patients dying within eight months of nephrectomy. Since chronic nephritis is a progressive and bilateral condition, it is only to be expected that the mortality rate should be high. In none of these cases was there anything to indicate the true nature of the pathologic process before operation.

URETER

Anomaly.—Crenshaw [26] added new cases to those reported by him in 1935. He reported 6 cases of aberrant ureter with extravesical orifices (bringing his total to 13 cases); 3 new cases of abortive ureteral development (totaling 5 cases), and a second case of crossed ureteral ectopia. Another case of multiple calculi in an extrapelvic cystocele is reported.

Crenshaw summarized certain data concerning the 13 cases of aberrant ureter with extravesical orifices. In 12 of the cases the patients were females. Four were children. In all but 1 case the pelvis was duplicated, and all the aberrant ureters came from the upper part of the pelvis. There was 1 case of bilateral duplication with bilateral extravesical orifices, necessitating bilateral heminephrectomy. The excretory urogram does not readily outline the abnormal pelvis, frequently because of reduced renal function in the anomalous upper segment of the kidney; instead, dependence on an elongated renal shadow with a disproportionate amount above the upper calix of the lower segment is significant. Retrograde pyelographic examination of the lower segment cannot always be depended on. Urethroscopic examination may reveal the aberrant orifice, but this orifice is not always easy to find and is not necessary for establisment of a diagnosis. Cystoscopic examination should be made to determine that only the two normal ureteral vesical orifices are present. Heminephrectomy resulted in cure in all cases.

---

26. Crenshaw, J. L.: Ureter with Extravesical Orifice: Supernumerary Ureter Ending Blindly; Crossed Ureteral Ectopia; Stones in Extrapelvic Cystocele; Report of Eleven Cases, Tr. Am. A. Genito-Urin. Surgeons **32**:133-155, 1939; J. Urol. **43**:82-101 (Jan.) 1940.

The 3 new instances of abortive ureteral development were incidental discoveries encountered during study of patients who were undergoing cystoscopic or urographic examinations for other reasons. No patient required any treatment.

In the second instance of crossed ureteral ectopia the patient was an 8 year old boy who had numerous other congenital deformities. Urologic studies revealed a solitary (right) kidney opening into the left side of the bladder by a dilated ureter. The right ureteral orifice was not present. Nephrostomy on the right side relieved the patient of his symptoms.

A cystocele following hysterectomy prevented complete emptying of the bladder in a 54 year old woman. Vaginal repair with vaginal cystostomy and removal of calculi resulted in satisfactory recovery.

Calculi.—Ormond [27] stated that ureteral calculus in each case presents the following questions: Is immediate operation desirable? If not, should manipulation be tried? If so, what method should be used? How long is it safe to temporize?

Each surgeon must be guided by his own experience, judgment and skill. Ormond has come to the conclusion that only the less drastic forms of manipulation are indicated. Patients who have stones the expulsion of which cannot be stimulated by gentler measures should be operated on after a sufficient period has passed for the stones to be expelled. Such a period will be measured by the amount of disability caused by the pain and by careful evaluation of the damage produced. Ormond has seen a stone lodged in the lower part of the ureter expelled after six months of careful observation. This stone caused in that time only four days of disability to the patient and no appreciable damage to the kidney. Some patients treated by multiple manipulations have been subjected to more danger, have suffered more pain and have lost more time than if they had been operated on in the first place.

In all cases, whether operation is done or not, attempts to prevent recurrence should be made. Ureteral dilation, pelvic irrigation, treatment of infection and attention to diet, to fluid intake and to changing urinary reactions usually will prevent recurrence.

Thompson and Kibler [28] reported 361 cases in which ureteral calculi were manipulated transurethrally. Approximately an equal number of patients were treated expectantly, and a similar number (392) were treated surgically. Cases in which simple diagnostic ureteral catheterization was carried out were not included.

---

27. Ormond, J. K.: Complications and Dangers of Lower Ureteral Calculi, Surg., Gynec. & Obst. **70**:584-587 (Feb., no. 2A) 1940.

28. Thompson, G. J., and Kibler, J. M.: Treatment of Ureteral Calculus, with Particular Reference to Transurethral Manipulation, J. A. M. A. **114**:6-12 (Jan. 6) 1940.

Anuria and uremia are the only indications for emergency treatment. Time and sedation will relieve many patients of their stones, but the type of treatment employed must be individualized. Whether manipulation should be done depends on the size of the stone, the size of the ureter, the position of the stone in the ureter and ready access to the ureter from below. Manipulation should be performed in the hospital with the patient under anesthesia. Manipulation usually is done through either the Braasch cystoscope or the McCarthy panendoscope by the following instruments: (1) multiple catheters, (2) Councill extractor, (3) the Johnson extractor, (4) a spiral stone dislodger and (5) the Collings knife. Postoperatively, two ureteral catheters are passed to the pelvis and left in place for forty-eight to seventy-two hours to obviate development of obstructive edema. Roentgenograms made postoperatively will demonstrate whether all of the stone has been removed.

In 330 cases (91.4 per cent) the results were immediately or subsequently successful. In 4 cases (1.1 per cent) the result was complete failure. In 27 cases (7.5 per cent) elective or essential surgical treatment was administered for removal of the stone after attempted manipulation. An average of 1.26 manipulations was necessary for each case in which the procedure was successful.

Serious fever developed in 2.7 per cent of patients after removal of the inlying ureteral catheters. These patients responded to reinsertion of the catheters or to the intravenous administration of mercurochrome (the disodium salt of 2.7 dibromgammahydroxymercurifluorescein). In 8.8 per cent of the patients mild reactions occurred. Severe complications were rare. One patient died after manipulation and surgical treatment, death being referable to a massive pulmonary embolus which developed while the patient was satisfactorily convalescent. A short period of hospitalization is one of the main advantages of transurethral removal of ureteral calculi; 75 per cent of the patients remained in the hospital less than one week. Failures and complications cause an occasional prolonged stay. The economic factor should not cause the surgeon to try too forcefully to succeed. Absence of evidence of renal function on the affected side is valueless in the presence of an impacted ureteral calculus. Excretory urograms taken several weeks later may show a normal pelvic outline.

O'Conor [29] discussed prostigmine methylsulfate as an aid in the expulsion of ureteral calculi.

The original dosage recommended by Hager (1.0 cc. of a 1:2,000 solution [0.5 mg. of the active substance injected subcutaneously] of

29. O'Conor, V. J.: Prostigmin as an Aid in the Expulsion of Ureteral Calculi, Tr. Am. A. Genito-Urin. Surgeons **32**:185-191, 1939.

prostigmine methylsulfate at three to four hour intervals for four doses) is still the most satisfactory. O'Conor used this dosage in the treatment of 52 patients. Seventeen of these patients received from two to four series of injections. The selection of patients for nonsurgical treatment was made with the usual recognition of the problem presented by each patient.

In 16 cases ureteral meatotomy, ureteral dilation and insertion of multiple catheters or bougies were employed, with the injection of sterile olive oil, papaverine hydrochloride and the like, without dislodging or producing passage of the stone. In the knowledge that the lower part of the ureter was capacious and in the absence of complications which would call for surgical intervention, prostigmine was administered, with resultant rapid downward progress or expulsion of the calculus in 12 of the 16 cases. In 9 cases the calculi passed within twenty-four hours after the injection of prostigmine methylsulfate. In 2 patients a second course of injections administered after a three day interval resulted in expulsion of the stone in eight hours and fourteen hours, respectively. One patient passed three large calculi after a third course of prostigmine methylsulfate, no cystoscopic treatment having been administered for three months. The calculi had remained in the same position, as determined by roentgenograms, for a period of eighteen months.

Two patients passed multiple calculi after receiving injections of prostigmine methylsulfate when both manipulative and surgical attempts at removal had been unsuccessful. During the period of this survey (twenty-three months) 13 patients were relieved of ureteral calculi by operation, and for 1 of the aforementioned patients operation was unsuccessful. Seven other patients were treated successfully without resort to injections of prostigmine methylsulfate. Thus, in summary concerning 66 patients treated, prostigmine methylsulfate was used as an additional aid in the treatment of 52, and in 18 of these the clinical response was so direct and so obvious as to establish in O'Conor's mind, without any reasonable doubt, the virtue of this medicament.

Injury.—Hyman and Wilhelm [30] reported 5 cases in which ureteropelvic anastomosis was established after avulsion, with a discussion of the procedures necessary for successful results in such establishment.

They stated that reimplantation of a ureter severed from the renal pelvis, either accidentally or in the treatment of obstructive hydronephrosis, is based on the following principles: (1) provision of free urinary drainage by means of nephrostomy, (2) accurate suture of the

30. Hyman, A., and Wilhelm, S. F.: Ureteropelvic Anastomosis Following Avulsion, J. Urol. **43**:52-60 (Jan.) 1940.

ureter to the pelvis without tension and (3) splinting of the anastomosis with a catheter to immobilize the ureter and to maintain the stoma.

It is generally agreed that nephrostomy should be performed in all cases in which any extensive plastic procedure is done on the pelvis or the ureter. The tube acts as a safety valve, preventing undue internal tension on the plastic suture lines. Free drainage is the first requisite in the treatment of urinary infection, from which most of the patients under consideration suffer.

On the basis of their experience with plastic operations on the pelvis for obstructive hydronephrosis as well as their experience in cases of ureteral avulsion, the authors expressed the belief that prolonged drainage is often desirable.

Anastomosis between the ureter and the pelvis should be established with accurate apposition of the parts. When possible, it is preferable to suture the mucosa and the muscularis in separate layers and to use fine interrupted plain catgut sutures for the mucosa and fine interrupted chromic sutures for the outer layers.

A straight splinting ureteral catheter, inserted into the severed ureter for about 10 cm. and brought out through the renal pelvis and cortex, facilitates anastomosis. It also serves the double purpose of splinting the ureter and maintaining the stoma. Although this catheter is a foreign body, its advantages appear greatly to outweigh its drawbacks. Too early removal of this catheter may result in stricture or in complete occlusion of the stoma. The splinting catheter should be left in place for at least ten days.

Late stricture not uncommonly follows reimplantation of a severed ureter into the pelvis. Frequent examinations should be done by excretory urographic methods. Any narrowing at the ureteropelvic junction is an indication for prompt cystoscopic dilation.

Transplantation.—Lower [31] stated that transplantation of the ureters into the rectum or the sigmoid flexure in the presence of the congenital deformity exstrophy of the bladder has been practiced for many years. The length of time for which a child could live in comfort after undergoing such a procedure before dying from infection in the upper part of the urinary tract did not seem of as much concern as the comfort such a child would enjoy during life and the lessening of the care he would need. It soon became apparent that a child would probably live as long after this procedure as if no surgical treatment for exstrophy had been given, if not longer. Sufficient time has now elapsed in a series of cases large enough to permit evaluation of this procedure and to enable

31. Lower, W. E.: Transplantation of the Ureters into the Rectosigmoid and Cystectomy: Motion Picture Demonstration, Tr. Am. A. Genito-Urin. Surgeons **32**:163, 1939.

urologic surgeons to apply it to the treatment of other conditions, such as malignant processes in the urinary bladder, intractable interstitial cystitis, tuberculosis of the bladder and (in certain instances) vesicovaginal fistulas.

Lower exhibited a motion picture of a technic which he has adopted and which has simplified the procedure and reduced the operative mortality rate. To the date of his report, his series consisted of 105 cases, the operation having been performed for various anomalies and pathologic conditions of the urinary bladder. He followed the patients and found that a majority of those living were entirely free from symptoms and that renal function was good even after many years had elapsed. One woman became pregnant and was delivered of a healthy child by cesarean section three years after undergoing ureteral transplantation; other patients have carried on their various vocations and have worked in perfect comfort.

Intussusception.—Bumpus [32] reported a case in which intussusception of the ureter afflicted a man 52 years old. The renal stone was removed, after which the patient experienced further trouble. Urograms and ureteral catheterization revealed what appeared to be intussusception of the ureter, a condition which Bumpus construed to be caused by a fragment of stone which had remained in the ureter. The ureterogram showed definite intussusception of the lower segment of the ureter into the upper. Subsequent passage of the stone and disappearance of ureteral torsion and narrowing confirmed this view. This case is of interest for two reasons: first, because of the uniqueness of the intussusception and, second, because of the spontaneous disappearance of the ureteral torsion as periureteritis subsided.

Hemorrhage.—Hamer [33] reported a case in which fatal ureteral hemorrhage was caused by erosion into the iliac artery, the erosion occurring during drainage by indwelling catheter for pyelitis of pregnancy.

The patient was a woman 18 years old and five months pregnant. She had bilateral pyelonephritis, an elevated temperature and chills. Her condition was relieved by the insertion of ureteral catheters, which were left in place. The catheters were replaced three times in three weeks. On the day before the patient died, sudden hemorrhage caused her to lose a large amount of blood. In spite of performance of the usual necessary measures, the patient died. Postmortem examination

32. Bumpus, H. C., Jr.: Intussusception of the Ureter, Tr. Am. A. Genito-Urin. Surgeons **32**:127-131, 1939.

33. Hamer, H. G.: Fatal Ureteral Hemorrhage Due to Erosion into the Iliac Artery: Report of a Case Occurring During Indwelling Catheter Drainage for Pyelitis of Pregnancy, Tr. Am. A. Genito-Urin. Surgeons **32**:177-183, 1939.

showed the right ureter to be attached to the common iliac artery at the pelvic brim, and an opening 1 cm. in diameter connected these structures at this point. The kidneys contained abscesses in the parenchyma and in the cortex. The right renal pelvis and ureter were full of blood clots.

This case was somewhat similar to those reported by Davidson and Taylor.

Hamer reported another case, in which a woman 31 years old and four months pregnant exhibited evidence of infection of the urinary tract. A ureteral catheter was inserted and was changed ten times during the following three months. The patient had marked anemia and received a number of transfusions of blood. Later she gave birth to a premature child and died. Necropsy showed that communication had existed between the external iliac artery and the right ureter.

The cause of the perforation in Hamer's case was fairly well established by the pathologist's report, yet there is reason for speculation as to the contributing cause, which may have been one of the following factors:

1. Use of too large and too rigid catheters.

2. Repeated injury to the ureter in one situation as the result of numerous insertions of catheters.

3. Pressure of the head of the fetus on the ureter containing the catheter so that the ureter was pressed against the vessel, the constant pulsation of which weakened the ureteral and vascular walls at the point of crossing.

4. Changes in the ureteral wall incident to pregnancy.

Paschkis, in the discussion of a case of perforation of the ureter by an impacted calculus, remarked that such an occurrence, although infrequent, is not extraordinarily rare. He questioned the supposition that the ureter ruptured at a site at which its wall was normal and expressed the opinion that manipulation during catheterization might have led to perforation in the particular case he was discussing.

Dilatation.—Franche [34] called attention to a process which is the reverse of the well known repercussions of ureteral lesions on the renal pelvis and on the kidney, namely, an important influence of the pelvis and the kidney on the ureter, resulting in dilatation of the latter for which no adequate cause exists in the lower part of the urinary tract and which can be explained only on the basis of the existence of a pathologic condition of the kidney or of the renal pelvis, most commonly hydronephrosis or renal ptosis. Such dilatation may involve the entire

34. Franche, O.: Dilatation urétérale au cours des lésions pyélo-rénales, J. d'urol. **46**:401-414 (Nov.) 1938.

ureter or may be localized in its upper two thirds, extending as far as its point of entrance into the lesser pelvis. The influence is not constant, however, and, although the dilatation may be considerably advanced, it does not necessarily affect the ureter, which may remain entirely normal in the presence of enormous hydronephrosis. There is no parallelism between the extent of the pyelorenal pathologic process and the degree of ureteral dilatation present.

It has been customary to believe that the changes under consideration are caused by pregnancy or that, at least, they have a genital origin. But the history of a woman who had never been pregnant and who had no history of involvement of the genitalia but who had a movable kidney would suggest that the cause lay in renal ptosis itself. Such an interpretation also seems to explain another fact that pregnancy has not explained, namely, the predominance of postgravidic sequelae on the right side, whereas it is well known that ureteropyelic dilatation during pregnancy is bilateral. It is known, however, that renal ptosis is more common on the right side in female patients, and this fact seems to throw some light on the cause of the ureteral dilatation under discussion. Additional confirmatory evidence is found in the fact that dilatation of this kind is found at times in men suffering from renal ptosis. A case was cited by Franche in which nephrectomy was about to be performed in some other surgeon's service, when Marion, being called in, advised that a pyelogram with the patient in the vertical position be made first, whereupon it was revealed that fixation of the kidney and not nephrectomy was needed. Nephropexy brought immediate relief, which had persisted for two years at the time of Franche's writing, the ureteropyelographic image at that time being perfectly normal. Two additional cases were described to show that such cases are not exceptional. Still other cases were presented, revealing that when the cause of ptosis or hydronephrosis has been removed ureteropyelic dilatation markedly diminishes and painful symptoms disappear.

In cases in which the condition might be considered "reversible," as has been explained, it would appear that the mechanism of production of ureteral dilatation can be nothing other than a state of ureteral atony, which in the course of time may result in permanent and definitive modification of the ureteral wall, a modification which in the earlier stages was transitory and reversible. It has been suggested that this atony is reflex, possibly caused by irritation of a nerve center or of a neuromuscular center situated in the upper part of the ureter, where it might be produced by a kink, renal ptosis, the presence of an abnormal vessel or some similar anatomopathologic condition. In the present state of urologic knowledge this explanation seems the most plausible that can be reached.

Gloor [35] compared cases of megaloureter without obstruction of the urinary flow and cases of marked stenosis of the ureteral outlet in which only slight dilatation of the ureters is exhibited. He opposed the opinion that mechanical obstruction causes megaloureter and advocated Bard's theory that dilated structures similar to the ureter in the presence of megaloureter originate as a result of dysplastic changes in the walls of such structures. Gloor found no proof that inflammatory changes of the ureteral wall alone could cause large dilatation of the ureter.

PROSTATE

General Considerations.—Wildbolz [36] discussed, in a paper read before the German Surgical Association, the treatment of benign hypertrophy and carcinoma of the prostate gland. The development of the normal prostate gland, the mechanism of prostatic hypertrophy and carcinoma of the prostate were discussed. He also reviewed and criticized the theory of hormonal influence. In another part of the paper, Wildbolz considered the operative and nonoperative methods of treatment: glandular therapy, operations on the genitalia (Steinach), high voltage roentgen therapy, suprapubic and perineal prostatectomy and transurethral prostatic resection. On the basis of Wildbolz' wide experience, he is especially able to discuss all these various methods of treatment. He expressed particular favor for perineal prostatectomy, which, he stated, is the only method which may produce a permanent cure of carcinoma of the prostate gland.

Kraas [37] reviewed results obtained in the various German surgical clinics in instances of transurethral resection of the prostate gland. He stated that there is a wide divergence of opinion as to the merits of transurethral resection and the open operation. He expressed the opinion that both methods have their place and that the operation should be suited to the particular condition of each patient.

Hypertrophy.—Deming [38] stated that the early phases of benign prostatic overgrowth are found in the muscular walls of the posterior portion of the urethra in specimens observed at necropsy.

Benign prostatic overgrowth usually passes through two phases of development. The first phase is the development of a fibromuscular

35. Gloor, H. U.: Ueber die Ursachen der Megaloureterbildung, Schweiz. med. Wchnschr. **69**:1080-1084 (Nov. 4) 1939.

36. Wildbolz, H.: Die Behandlung der Prostatahypertrophie und des Prostatacarzinoms, Zentralbl. f. Chir. **66**:770-782 (April 8) 1939.

37. Kraas, E.: Prostatektomie—Prostataresektion, Ztschr. f. Urol. **33**:553-559, 1939.

38. Deming, C. L.: The Development of Prostatic Hyperplasias, Surg., Gynec. & Obst. **70**:588-594 (Feb., no. 2A) 1940.

mass from the intramuscular stroma of the posterior part of the urethra. The second phase is invasion of the fibromuscular nodule by the epithelium of a prostatic duct. This epithelial proliferation develops prostatic glands and ducts of normal appearance.

The epithelial element overgrows the fibromuscular element so as to make the full-grown lesion appear glandular. The glands in the posterior part of the urethra and the prostate gland are not primarily involved.

The primary fibromuscular nodule resembles fibromyoma of the uterus and may be derived from a remnant of the musculature of the müllerian ducts.

Benign overgrowth of the prostate gland produces hyperplastic tissue and not hypertrophied tissue.

Castration in Cases of Prostatic Hypertrophy.—Huggins and Stevens,[39] in discussing castration and benign prostatic hypertrophy, stated that in 50 cases of benign prostatic hypertrophy the prostatic epithelium varied from flat to tall cylindric, the latter type being found in every case.

From 3 men who had benign prostatic hypertrophy, specimens of prostatic tissue for biopsy were obtained at the time of castration and also twenty-nine, eighty-six and ninety-one days later. Epithelial atrophy was not present twenty-nine days after castration but appeared plainly at eighty-six and at ninety-one days after the operation. In 1 case there were a marked reduction in the size of the prostate gland at rectal examination and an increase in the size of the urinary stream within one month after castration. The evidence derived from castration concerning benign prostatic hypertrophy in man supports the view that the prostatic epithelium, at least, is under control of the testes.

Total Perineal Prostatectomy.—Lowsley [40] stated that total perineal prostatectomy should be done more often than it is at present. It is indicated in cases of early carcinoma, chronic pyemia, intractable chronic fibrosis and calculosis and in certain cases of tuberculosis and adenoma of the prostate gland. He suggested that the membranous portion of the urethra be joined to the bladder with a mattress suture of ribbon gut; this procedure not only makes the urethra continuous with the bladder but plicates the external sphincter and prevents incontinence of urine. The success obtained in the cases in which the operation was done encouraged Lowsley to report the method in the hope that others would use it.

---

39. Huggins, C., and Stevens, R. A.: The Effect of Castration on Benign Hypertrophy of the Prostate in Man, J. Urol. **43**:705-714 (May) 1940.

40. Lowsley, O. S.: Total Perineal Prostatectomy, J. Urol. **43**:275-285 (Feb.) 1940.

Vest [41] has introduced a modification of the conventional radical perineal prostatectomy of Young for carcinoma of the prostate gland. He sai l that this modification diminishes the liability to incontinence, which frequently follows the operation because of injury to the external urinary sphincter by the sutures used to approximate the margin of the bladder to the membranous portion of the urethra. In the performance of this anastomosis it becomes necessary at times to take sutures well t rough the triangular ligament, an action which must inevitably include and injure the external sphincter.

To avoid this, Vest, in uniting the bladder to the membranous portion o the urethra, has used three mattress sutures, which have been placed in the margin of the anterior part of the bladder, traverse the external sphincter on either side of the urethra just beneath the mucosa, continue just beneath the mucosa through the region of the sphincter through the triangular ligament and pass into the perineum just beneath the skin on the anterior margin of the perineal incision. When traction is made on these three sutures and when they are tied, a considerable part of the circumference of the cut margin of the bladder is drawn tightly against the membranous portion of the urethra and anchored there without any interference with or constriction of the sphincter in this region. The remainder of the bladder is then closed, and a figure-of-eight is used to complete the union of the bladder to the under margin of the membranous portion of the urethra.

Vest has used this modification in four operations, with satisfactory results.

Transurethral Resection.—Tolson [42] reviewed the records of 356 patients operated on for relief of obstruction to the vesical outlet from 1924 to 1938, inclusive. One hundred and forty-four patients were operated on by means of modifications of the punch technic. During the period from 1932 to 1936, 96 patients were operated on by means of electrical resection. One hundred and sixteen patients were treated by suprapubic prostatectomy. For the entire period under discussion, the approximate ratio of resection to prostatectomy was 2 to 1. During 1938, resection was performed in 56 instances and prostatectomy in 4, a ratio of 14 to 1.

No patient less than 50 years of age was subjected to prostatectomy, whereas 6 patients less than 50 years of age underwent resection. In this group the obstruction was minor or moderate. Ten patients more than 80 years of age underwent resection. These patients had advanced degrees of prostatic enlargement.

41. Vest, S. A.: Radical Perineal Prostatectomy, Surg., Gynec. & Obst. **70**: 935-937 (May) 1940.

42. Tolson, H. L.: Prostatic Resection with Modifications of Young's Punch, J. Urol. **43**:116-122 (Jan.) 1940.

The type of anesthesia employed for patients who underwent resection was low spinal anesthesia produced by procaine hydrochloride for 134, caudal anesthesia produced by procaine hydrochloride for 90 and local anesthesia produced by procaine hydrochloride for 16. Low spinal anesthesia induced by procaine hydrochloride is the anesthesia of choice; the usual dose is 120 mg.

During 1938, 56 patients underwent resection. Forty-two patients underwent removal of less than 20 Gm. of tissue and 14 patients underwent removal of more than 20 Gm. of tissue. The maximal quantity removed at one operation was 69 Gm.; the average quantity was 18.1 Gm.

Twelve patients in whose cases preoperative diagnosis of prostatic carcinoma had been made underwent resection. In these instances there was obstruction to urination which demanded relief. The patients were treated postoperatively by high voltage roentgen therapy.

Patients who underwent resection by cold sharp steel experienced fewer complications than did patients who underwent resection by the electrical method. The problem of infection is less annoying, and the period in which pyuria is present is shorter, when the former technic is employed. Prolonged pyuria and prostatitis occurred in a large percentage of the patients who underwent electrical resection and infrequently in those who underwent cold resection. Prostatitis following cold resection responded more promptly to treatment.

Extravasation of urine occurred in 2 patients, but it was immediately recognized and treated by suprapubic drainage. Overdistention of the bladder with irrigating fluid was the probable cause of this complication. For some time it has been Tolson's custom not to drape the lower portion of the abdomen; this permits his assistant to note the degree of distention of the bladder.

Epididymitis occurred in 5 patients who had not undergone vasectomy. Vasectomy has been used for the majority of patients who undergo resection, and in no instance has epididymitis occurred after vasectomy. A considerable number of patients have objected to being sterilized. Two patients treated by resection, aged 69 and 72 years, subsequently became fathers of additional members of their families.

Ten deaths (4.1 per cent) occurred in the group of 240 patients treated by transurethral resection. In the group of 116 patients treated by suprapubic enucleation there were 9 deaths (7.7 per cent).

Removal of the obstructing portion of the prostate gland by sharp cold steel, with coagulation of only the bleeding points, is accompanied by minimal injury to the residual portion of the prostate gland. Early epithelization of the prostatic portion of the urethra is rendered possible by the presence of a freshly cut surface composed, except for the small, lightly coagulated points, of living cells. Consequently, convalescence is relatively short, and there is minimal morbidity.

Foley [43] stated that the status of the surgeon who does transurethral resection determines the status of prostatic resection in his hands but not in general. Great competence of such a surgeon makes prostatic resection a valuable procedure to be used extensively, the asserted upper limits of the procedure's utility being 80 to 95 per cent of cases. Limited competence of the surgeon makes prostatic resection a procedure of limited value and restricted use; the asserted extent of its successful use in the hands of such surgeons is 15 to 50 per cent. For the completely incompetent urologic surgeon who uses the procedure, no gland is small enough and no transurethral operation is sufficiently foolproof to make resection the operation of choice. In such hands, if the surgeon possesses any surgical ability whatever, open operation is the operation of choice. Skill, ability and experience on the part of the surgeon who does transurethral resection extend the limit set by the size of the gland and the limit of technical difficulty within which resection is the operation of choice.

Suprapubic and perineal prostatectomy are the same perfected and useful operations that they were before any one ever heard of resection. They yield nearly perfect results, with a low mortality rate. Neither of them makes anything like the demand made by resection on special talent, long training in cytoscopy and experience with the procedure itself.

The process of development and perfection of suprapubic and perineal prostatectomy entailed poor results and a mortality rate that now seems shocking. There was no alternative. Perfected technic, modern surgeons well skilled in the procedure of open prostatectomy and what they contribute to human welfare have vindicated the effort. The process of development of transurethral prostatic resection to its present stage of perfection likewise entailed poor results and a mortality rate that now seem shocking. Fortunately, reversion to major prostatectomy was not the only alternative for all urologic surgeons who did transurethral resection. A perfected technic of resection and urologists who are skilled in the procedure of resection now vindicate the effort, and it remains only for the urologic surgeon who uses transurethral resection to determine his status.

The surgeon who continues routinely to use either suprapubic or perineal prostatectomy on the ground that in his hands it is the operation of choice and that in his hands it best serves the welfare of the patients is fair to the patient, to medical science and to himself.

Urologists and general surgeons alike must limit their use of resection according to their ability to perform transurethral resection, and

43. Foley, F. E. B.: The Present Status of Transurethral Resectionists, Competent and Otherwise, J. Urol. **43**:565-571 (April) 1940.

they must always employ the operation that best serves the welfare of the patient in their hands, whether it be by the suprapubic, the perineal or the transurethral method. It is desirable to be able to use each procedure with skill, ability and reasonable competence, but shortcomings in respect to any of the three procedures are no reflection on that procedure, nor do they enhance the value of the others. If this is recognized and if surgeons are governed accordingly, prostatic operations will be perfectly individualized in respect to both the patient and the surgeon, and urologic surgeons will best serve the welfare of their patients.

Carcinoma.—Silverstone [44] stated that radium has a definite place in the treatment of carcinoma of the prostate gland. To be of maximal benefit, irradiation should be uniform, and, since massive local destruction is to be avoided, multiple foci of low intensity should be used. Perineal exposure of the prostate gland allows a uniform distribution of needles, without concentration of the dose at any mucosal site. Neoplastic extension in the neighborhood of the seminal vesicles can also be dealt with; if marked sepsis or renal inefficiency is present, such extension must be dealt with by preliminary drainage, either by indwelling catheter or by suprapubic cystostomy.

Radium is not universally applicable. The patient who has distant metastases and pain referred to the lower limbs, usually caused by glandular metastases in the pelvis, will be better without treatment by radium. About a third of patients have demonstrable metastases when they are first seen. If the general condition is poor or if the kidneys are inefficient, radium should not be used unless and until improvement takes place. When marked obstruction is present, conservative surgical intervention usually should be carried out in addition to radium therapy. Patients treated with radium should be followed up carefully, since if fibrosis occurs dilation or endourethral resection may be necessary.

For treatment to be of value in cases of carcinoma of the prostate gland the condition must be diagnosed earlier than is usually the case. It would seem that this can be accomplished only by the routine rectal examination twice a year of persons more than 50 years old. Such examination may reveal areas of induration, the pathologic aspects of which can be confirmed by resort to aspiration of material for biopsy.

Sarcoma.—Stevens and Barringer [45] presented a paper concerning sarcoma of the prostate gland and anaplastic carcinoma. The latter is

---

44. Silverstone, M.: Radium in Treatment of Carcinoma of the Prostate, Brit. J. Surg. **27**:498-505 (Jan.) 1940.

45. Stevens, A. R., and Barringer, B. S.: Sarcoma of the Prostate, Tr. Am. A. Genito-Urin. Surgeons **32**:275-303, 1939.

readily and commonly confused with sarcoma. The purpose of the paper was to correlate existing facts and thus perhaps to clarify ideas concerning these diseases.

Brief histories were appended of 16 patients who had prostatic neoplasms. Five of the tumors were proved microscopically to be myosarcomas, 1 to be lymphosarcoma and 1 to be spindle cell sarcoma. There were 3 carcinomas in young men; for the last 6 patients a clinical diagnosis only of malignant disease had been made, the lesion being called sarcoma. It is of interest to note that 4 of these patients were subjected to operation for prostatic abscess. According to the literature, only in rare cases can sarcoma of the prostate gland be controlled for more than a few years by present methods. On the basis of a review of the histories, the records are discouraging. The diagnosis is usually made late in the course of the disease, and the course is rapidly downward, with occasional temporary regression of the tumors if the patient is subjected to roentgen therapy.

The tissue giving origin to the tumor forms the basis for perhaps the simplest and most satisfactory pathologic classification of prostatic neoplasms. Stevens and Barringer, therefore, suggested that sarcomas of the prostate gland be segregated in three groups:

1. Leiomyosarcoma and rhabdomyosarcoma, which take origin in the musculature of the prostate gland. These two tumors are much alike in growth and clinical manifestations.

2. Lymphosarcoma, which originates in the lymphatic tissue of the prostate gland. This tumor formerly was a disputed entity but is now generally recognized.

3. Sarcoma, which has an undetermined origin. The term includes spindle cell sarcoma, fibrosarcoma, myxosarcoma, round cell sarcoma and giant cell sarcoma.

For clinical reasons, Stevens and Barringer added to the aforementioned groups a fourth lesion, which is not sarcoma but anaplastic carcinoma. It has various features which resemble those of sarcoma and is often mistaken microscopically for lymphosarcoma.

Metastasis from carcinoma of the prostate gland takes place usually through the lymphatic channels but may progress by means of the blood vessels. It is probable that the metastasis to bone occurring in this disease may take place by either the lymphatics or the blood vessels. The picture is reversed in cases of sarcoma of the prostate gland, in which the usual routes of metastasis are the blood vessels. Lymphatic metastases from prostatic sarcoma are not rare, however.

The less malignant sarcomas often cannot be distinguished from nonmalignant hypertrophy of the prostate gland. When the diagnosis is made on the basis of the specimen removed, any future treatment depends on the pathologist's opinion and the subsequent course of the disease. The more malignant tumors, whether they are lymphosarcomas, myosarcomas, sarcomas of undetermined origin or anaplastic carcinomas, generally produce one or more indications that an unusual neoplasm is present. These indications are: The patient is below the age at which prostatic hypertrophy or prostatic carcinoma might be expected to appear; he gives a short history of the development of the lesion or his discomfort; the prostate gland is of great size; the neoplasm often almost fills the rectal lumen; the neoplasm is elastic and irregular as contrasted to the irregular indurations of prostatic carcinoma; a suprapubic tumor is present which is continuous with the prostatic tumor; generalized lymphadenopathy is present, and the patient has experienced rapid loss of weight and cachexia, although the two symptoms last mentioned generally are absent.

Prostatic abscess, prostatic carcinoma, tumor of the bladder, extravesical tumor, cyst of the prostate gland and massive stone of the prostate gland are all conditions which may be mistaken for prostatic sarcoma.

Stevens and Barringer's experience and the literature both demonstrate the superior value of irradiation over surgical procedures in the treatment of this condition.

Leiomyoma.—Deming [46] discussed the significance of leiomyomas of the prostate gland. "Leiomyoma" is a term loosely applied to tumors of smooth muscle and connective tissue. Deming found in the literature 32 cases of leiomyoma of the prostate which were available for study. The condition of the youngest patient, who was 24 years of age, was described in a case reported by Koenig in 1936; the condition of the oldest patient, who was 80 years of age, was described by Patch and Rhea in 1935. The average age is 61.7 years.

The cases are sharply divided into two groups. Cases in which the patients had obstruction of the neck of the bladder simulate in every way those cases, familiar to urologists, which are described as cases of "prostatism." Reports of necropsy showed that obstruction of the neck of the bladder, infection of the urinary tract, distention of the bladder and dilatation of the ureters and renal pelves were present, and in this particular group there was a majority of 27 cases. The average age of the patients was 64.7 years, approximately the same as that

46. Deming, C. L.: The Significance of Leiomyomata of the Prostate, Tr. Am. A. Genito-Urin. Surgeons **32**:263-273, 1939.

for patients with prostatic hypertrophy. The second group of patients had rectal symptoms without urinary symptoms. Rectal examination showed a smooth, elastic enlargement of the prostate gland. There were 5 patients in this group, and the average age was 45.2 years.

In the series of 32 cases, 5 patients died without undergoing operation on the prostate gland. Twenty patients were operated on by the suprapubic route. The youngest patient, 24 years of age, who had no urinary symptoms, was included in this operative group. All the patients recovered. Six were treated by perineal enucleation of the tumor. Five recovered; 1 died within twenty-four hours, of shock. Transurethral resection was performed in only 1 case; it was successful. Twenty-six of the 27 patients on whom operations were performed recovered, the operative mortality rate being 3.7 per cent, which approximates the results of prostatic operations in the best clinics.

The tumors varied in size from 5 to 570 Gm.; they occupied the urethral portion of the prostate gland.

Many authors have described leiomyomas as encapsulated tumors of smooth muscle, simulating myomas of the uterus. In other reports, leiomyomas have been described as nonencapsulated tumors of smooth muscle with interlacing fibers having a whorled appearance.

Up to the present, the diagnosis of leiomyoma of the prostate gland has not been made preoperatively. Rectal palpation does not distinguish leiomyomatous and glandular tumors. It is possible that the occurrence of rectal symptoms in young men might suggest the existence of such a neoplasm. Cystoscopically, leiomyomatous enlargements of the prostate gland are similar to the glandular masses in situation, size and character. A specimen for biopsy obtained by the transurethral method provides a means of diagnosis. This method was employed in only 1 reported case, but the lesion was not diagnosed until microscopic study had been made.

Abscess.—Schwartz [47] found, in studying the problem of prostatic abscess and nonsuppurative prostatitis, that the nonspecific hematogenous type of abscess is as common as that caused by the gonococcus. Of the 12 cases of nonspecific abscess reported, there was sufficient clinical evidence in 4 to warrant the designation of "metastatic" or "embolic" abscess of the prostate gland. The history of several of the other patients suggested that the abscess was secondary to a focus elsewhere, which had already subsided when the patient presented himself. It is justifiable to assume that metastatic abscess of the prostate gland occurs much more often than is generally appreciated.

47. Schwartz, J.: Metastatic Abscess of the Prostate Gland, J. Urol. **43**:108-115 (Jan.) 1940.

Prostatic abscess is not a serious lesion if it can be recognized in time and if it can be properly treated. A conservative policy is justifiable and is often of great value, but surgical drainage is imperative when there are unmistakable signs of a prostatic abscess which does not respond to reasonable nonsurgical measures. Recovery of the patient after surgical drainage is usually more prompt than when drainage has not been instituted.

(To Be Concluded)

Notices

SPECIAL FEATURES FOR COMING YEAR

It is proposed to continue the publication of symposiums during the coming year and, in addition, to publish review articles which will cover the advances in all fields of surgery. The first of these articles will be by Drs. Alton Ochsner and Michael DeBakey; it will be entitled "Carcinoma of the Lung" and will appear in an early issue. A paper by Drs. W. H. Cleveland and Waltman Walters, "Surgical Lesions of the Pancreas: A Review," will appear shortly thereafter. Other members of the Editorial Board will sponsor articles of this type, and the board feels that in this way the recent developments in all fields of surgery will be brought to the attention of the readers of the ARCHIVES.